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Edited by
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of New York

With the Collaboration of
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SIR W. WATSON CHEYNE, C.B., F.R.S. of London

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EXTIRPATION OF THE CHOROID PLEXUS OF THE LATERAL VENTRICLES IN COMMUNICATING HYDROCEPHALUS

BY WALTER E. DANDY, M.D.

OF BALTIMORE, MD.

(From the Department of Surgery, The Johns Hopkins Hospital and University)

No form of treatment, either medical or surgical, has yet a valid claim to the cure of a single case of hydrocephalus, except in those cases caused by tumor and relieved by tumor extirpation. But hydrocephalus is a curable disease. This is demonstrated by the not infrequent cases which have been cured spontaneously, though usually at a time when cerebral destruction has left the patient a hopeless imbecile. The reason for nature's successes is that the cause has been either circumvented or overcome. The reason for medical or surgical failures is that the cause has not been recognized. All forms of therapy have been entirely empirical. They have been directed toward the effect rather than the cause. They have lacked not only the etiology and pathology of the disease but even a knowledge of the circulation of cerebrospinal fluid before pathological changes have occurred.

Hydrocephalus should no longer be classified as an idiopathic disease. Its pathology and, in large part, its etiology are definitely established.[1] With the cause recognized, a rational form of therapy is indicated. I make this statement principally upon the results of our own investigations, which have been conducted in the past five years. These studies include the paths for the circulation of cerebrospinal fluid, the place and manner of formation and absorption of cerebrospinal fluid, the experimental production of hydrocephalus, the pathogenesis of many cases of hydrocephalus studied clinically by the phenolsulphonephthalein test, and the pathology of the various so-called types of hydrocephalus by post-mortem examination. The present communication is directed to the treatment based upon these observations.

A knowledge of the type of hydrocephalus is an absolute prerequisite to its treatment. The timeworn symptomatic classifications are incoördinate and confusing. The classification on the following page is presented, based upon the etiology and pathology of the disease:

[1] Dandy, W. E., and Blackfan, K. D.: Internal Hydrocephalus. An Experimental, Clinical, and Pathological Study. *J. Am. M. Assoc.*, 1913, lxi, 2216; *Am. J. Dis. Child.*, 1914, viii, 406; *Beitr. z. klin. Chir.*, 1914, xciii, 392. Internal Hydrocephalus, Second Paper. *Am. J. Dis. Child.*, 1917, xiv, 424.

The vast majority of cases of hydrocephalus are included in one of the two groups: (1) Communicating hydrocephalus; (2) obstructive hydrocephalus. The other types are rare. It will be noted that no separate subdivision has been made into internal and external hydrocephalus. It is a question whether external hydrocephalus ever really exists as a primary condition. It seems to be a secondary transformation of a primary internal hydrocephalus. This metamorphosis is probably due to a local or general atrophy of the cerebrum permitting escape to the exterior by these artificial channels. Because of the rarity of this condition, however, our facts are not sufficient to make a positive stand in this statement. It is conceivable, though not proven, that it may also result from a transfer of cerebrospinal fluid from the subarachnoid space to the subdural space in communicating hydrocephalus. The exact status of so-called external hydrocephalus is still in doubt. With this rare exception, nearly all hydrocephalus is internal; that is, the accumulation of cerebrospinal fluid is in the ventricles. It is internal for the very

- Hydrocephalus due to
  - Diminished absorption of cerebrospinal fluid
    - Communicating hydrocephalus (due to adhesions in the subarachnoid space)
    - Obstructive hydrocephalus due to
      1. Congenital atresias
      2. Adhesions — Acute / Chronic
      3. Tumors — Acute / Chronic
    - External hydrocephalus (?)
  - Increased production of cerebrospinal fluid
    - Acute hydrocephalus (increased fluid from inflammatory products in acute meningitis and in trauma)
    - Communicating hydrocephalus (due to occlusion of the vena magna Galeni)

good reason that cerebrospinal fluid forms in the ventricles and cannot reach the exterior (obstructive hydrocephalus); or at most it reaches only a small fraction of the external absorbing surface—the posterior cranial fossa and spinal canal (communicating hydrocephalus). In either case the fluid dams back at its source. If cerebrospinal fluid could reach the subarachnoid space over the entire exterior of the brain, it would be absorbed and hydrocephalus could not exist.

Another rare type of hydrocephalus is that due to thrombosis of the vena magna Galeni. Owing to insufficient collateral venous circulation, hydrocephalus then results from an increased production of cerebrospinal fluid by venous stasis, just as ascites often follows stenosis of the inferior vena cava. I have produced this type experimentally, but have seen no instance clinically. Only a few cases have been reported in the literature.

In acute meningitis of inflammatory origin there is undoubtedly an increased production of fluid from the products of the inflammation. It is questionable whether an increase of fluid by exudation should be classified

with true hydrocephalus. It is probably external as much if not more than internal, because the infection has a general but principally external distribution. It subsides with the decline of the infection and is not of practical import in considering the treatment of hydrocephalus. An increase of fluid also follows trauma to the brain in fractures of the skull. It is probably of vascular origin, and usually subsides rapidly.

Fundamentally the two main types of hydrocephalus—obstructive and communicating—are similar. Both are due to an obstruction in the cerebrospinal fluid circulatory system. In our series of cases in children the relative frequency is nearly the same. In the former the obstruction is in the ventricles and in the latter in the subarachnoid space. The only reason for subdividing hydrocephalus into groups is that the anatomical differences in the two types necessitate an entirely different operative procedure for the treatment of each.

In this paper only the treatment of the communicating type of hydrocephalus will be considered. A form of treatment for hydrocephalus with obstruction will be presented in a later communication. For a proper understanding of the basis for the operation herein proposed, a brief explanation of the underlying etiology and pathology is necessary.

The Etiology and Pathology of Hydrocephalus with Communication.—It is the communicating type of hydrocephalus which has caused all hydrocephalus to be considered idiopathic. Though numerous explanations have been proposed, no pathologic findings have been presented until recently. All the ventricles communicate with each other and with the subarachnoid space, and post-mortem examinations of the brain have heretofore revealed nothing to the naked eye. The reason for the negative findings has been an inadequate knowledge (1) of the post-mortem appearance of the normal subarachnoid space and the pia arachnoid membranes; (2) of the alterations produced by pathological changes; (3) of the relation of the subarachnoid space to the absorption of cerebrospinal fluid; and (4) the anatomy and physiology of the cerebrospinal fluid circulatory system.

The only satisfactory time to observe changes in the meninges is when the brain is being removed. Adhesions of an extensive nature will then be seen and divided, but later will show very little in the preserved specimen. In cases of communicating hydrocephalus the meninges will be opaque and thickened, the normal filmy pia arachnoid will be replaced by a firm, fibrous, adherent membrane. This will be especially noted in the cisternæ at the base of the brain. The adhesions are often so dense as to tear the brain during their liberation. It is the distribution or location of these adhesions, not their extent, which determines the production of hydrocephalus (Fig. 5). Adhesions encircling the midbrain where it passes through the incisura tentorii, will destroy all communication between the posterior and middle cranial fossæ and thereby eliminate the entire subarachnoid space over both cerebral hemispheres from participation in the absorption of cerebrospinal fluid. Hydrocephalus will invariably result from such a process. Adhesions which

close the cisternæ at the base of the brain will also produce hydrocephalus just as effectively.

The extraventricular cerebrospinal circulatory system may be compared to the trunk and branches of a tree—the cisternæ representing the trunk, the subarachnoid spaces over the cerebral hemispheres the branches. Obliteration of the cisternæ by adhesions is equivalent to transection of the trunk of a tree. All distal communication will be destroyed and hydrocephalus will follow. Large areas of adhesions may be present over the cerebral hemispheres with only local effects and with no effect upon the general cerebrospinal fluid circulation. This is analogous to the absence of a relationship between the destruction of branches and the life of a tree. The absorbing function of these local areas is easily compensated by the subarachnoid space over the remainder of the brain.

In each of our cases these adhesions have also obliterated either one or two of the three foramina (Luschka and Magendie) by which the ventricles and the subarachnoid space normally communicate (Fig. 5). One foramen remaining patent will maintain an adequate transfer of cerebrospinal fluid. Should the adhesions close all three foramina at the base instead of only one or two, an obstructive hydrocephalus would result. In short, communicating hydrocephalus is due not to a reduction in the avenues of communication between the ventricles and the subarachnoid space but to a blocking of the primary trunks of the subarachnoid space, thus preventing cerebrospinal fluid from passing to the branches. The distribution of cerebrospinal fluid is thereby limited to that small part of the subarachnoid space in the posterior cranial fossa and the spinal canal. It does not include any of the subarachnoid space over either cerebral hemisphere, which is by far the most important area for absorption.

Cerebrospinal fluid is absorbed from the entire subarachnoid space. Adhesions obstructing the main stem of the subarachnoid space therefore limit the absorbing area to a fraction of the normal, and the diminished absorption results in accumulation of cerebrospinal fluid—hydrocephalus.

We have spoken of adhesions almost exclusively as the cause of communicating hydrocephalus. It is conceivable that certain tumors filling or compressing the cisternæ may produce similar results. We have as yet no proof of this possibility. All of the cases observed by Dr. Blackfan and myself have had a meningitis, which has been both prenatal and postnatal. There is frequently a definite history of meningitis preceding the development of hydrocephalus, but this cannot always be obtained.

Studies with the phenolsulphonephthalein test enabled us to predict ante mortem the pathologic findings. By the dye it was possible to measure the amount of absorption from any region by the quantitative excretion in the urine. It was found that normally practically no absorption occurs from the entire ventricular system (less than 1 per cent. in two hours). All the cerebrospinal fluid is absorbed from the subarachnoid space; 40 to 60 per cent. being excreted in a two-hour interval, which has been adopted as an

arbitrary standard of time. In a large series of cases of communicating hydrocephalus the absorption from the subarachnoid space was found almost invariably to be only 8 to 10 per cent., or about one-fifth of the normal.

In view of the fact that experiments on animals have demonstrated that absorption of cerebrospinal fluid is from the entire subarachnoid space, the lowered absorption in communicating hydrocephalus must mean a reduction in the extent of the absorbing area and at a fairly constant location. The localization of the adhesions blocking the cisternæ or surrounding the mesencephalon harmonized the physiological and clinical tests and the pathological findings. The frequent history of an antecedent meningitis is additional confirmatory evidence, should such be necessary. The experimental production of this type of hydrocephalus by duplicating nature's pathologic processes, leaves no doubt of the etiological and pathological basis of communicating hydrocephalus. This experimental proof will shortly appear, together with that of the other types of hydrocephalus.

In operations upon the brain of cases of communicating hydrocephalus, one is impressed by the absence of fluid in the sulci. The sulci are practically obliterated, and only vascular lines separate the convolutions. If hydrocephalus were due to occlusion of such fanciful structures as stomata into the venous sinuses or the Pacchionian bodies, as has been proposed, one should expect distended subarachnoid spaces up to the points of obstruction. The absence of extracerebral cerebrospinal fluid proves the non-existence of any such mechanism, and indicates the lack of communication of the part of the subarachnoid space with the fluid-containing spaces; in other words, an obstruction must exist at some place nearer the origin of the cerebrospinal fluid.

The Diagnosis of Hydrocephalus with Communication.—Clinically, the two types of internal hydrocephalus are identical. Neither by the history nor the physical examination can they be differentiated. Frequently, the communicating type can be diagnosed by the large quantity of cerebrospinal fluid which can be obtained by lumbar puncture, but there is such great variability in the amounts of fluid obtained in both types of hydrocephalus that the results are frequently of little value. The differentiation between the two types of hydrocephalus can be simply, harmlessly and absolutely made by the phenolsulphonephthalein test.

One cubic centimetre of neutral phenolsulphonephthalein[2] is introduced into either lateral ventricle; a lumbar puncture is done one-half hour later. If communication exists, the dye will by that time have appeared in the spinal fluid. If an obstruction exists in the ventricular system, the spinal fluid will remain colorless. This test is the only way in which guess-work can be eliminated.

Previous Treatment of Communicating Hydrocephalus.—Because of the

[2] This solution has been specially prepared by Mr. H. A. B. Dunning, of Hynson, Westcott & Dunning. A serious reaction will follow the use of the ordinary phenolsulphonephthalein solution used for kidney studies.

great number of operations suggested, tried, and found failures, their consideration individually would require too much space and yield little of practical value. With rare exceptions, no attempt has been made to differentiate the two types of hydrocephalus. The usual treatments have been directed toward a disposition of the fluid, (1) either by transferring the fluid to the exterior of the brain, to the scalp, the retroperitoneal space, the peritoneal cavity, or some other space; or (2) by making a communication with some part of the venous system, either directly or by a vascular transplant; (3) in addition to periodically repeated lumbar and ventricular puncture, attempts have been made to establish a more or less continuous drainage to the exterior, always, of course, with death from secondary infection. Other attempts have been made to reduce the formation of cerebrospinal fluid by ligating one or both carotid arteries, by injection of irritants into the ventricles, compression of the head, etc.

All attempts to drain into body spaces are futile because the tissues wall off the fluid and soon cease to absorb it. Moreover, the openings into the spaces remain patent only temporarily. Puncture of the corpus callosum might at first sight appear to be an ideal procedure, but it is practically identical with punctures elsewhere. Fluid side-tracked by an opening of the corpus callosum or of other parts of the brain does not pass into the subarachnoid space, but into the avascular subdural space, where absorption is little if any greater than in the ventricles or in the scalp. Moreover, the opening in the corpus callosum or elsewhere closes in the course of a few weeks. Fluid can reach the *subarachnoid spaces* over the brain only through the normally designated distributing channels—the cisternæ. Permanent communication between the ventricles and the cisternæ can be maintained only through the fourth ventricle and by means of the foramina of Luschka and Magendie, or, in their absence, by openings artificially produced in this region.

The fate of vascular communications is similar. The opening into the veins, sinus or transplant functions but briefly. Such openings can hardly be expected to function properly, because they should handle in a few minutes all the fluid which is produced in several hours, and would be functionless the remainder of the time. Normal absorption of cerebrospinal fluid takes place slowly, by a process of osmosis through membranes and not into prepared openings or stomata.

Scientific Basis for Operative Treatment.—The logical treatment of any disease is the removal of the cause. In communicating hydrocephalus the treatment of the cause, that is, the removal or liberation of adhesions, is precluded by present surgical limitations. The area of adhesions is too extensive, too diffuse, and in a location inaccessible for operative interference. The ideal treatment would be restoration of the cisternæ through these adhesions, and even if reconstruction were possible reformation of the adhesions must always remain a possibility.

In view of these deterring factors, treatment of the cause must for the

present at least be deferred. From the results of experiments on animals a new and scientific form of treatment is suggested, by which it is hoped to circumvent the cause. This treatment aims to restore the balance between fluid production and fluid absorption by reducing the production of cerebrospinal fluid to a level where it can be absorbed. By experiments on animals, the results of which will appear shortly, the following facts have been established:

(1) If the foramen of Monro is occluded, a unilateral hydrocephalus results;

(2) But if the entire choroid plexus of this ventricle is removed at the time the foramen of Monro is occluded, this ventricle will be obliterated;

(3) Therefore, cerebrospinal fluid forms from the choroid plexus, and not from the ependyma.

(4) Following total occlusion of the aqueduct of Sylvius the development of hydrocephalus is greatly retarded by the extirpation of the choroid plexus of both lateral ventricles. In this experiment the cerebrospinal fluid, which is produced proximal to the obstruction, can be derived only from the choroid plexus of the third ventricle.

The conclusion from these experiments is if all the choroid plexuses of the four ventricles could be removed, the formation of cerebrospinal fluid would cease and hydrocephalus could not result or if present its development would cease. To cure obstructive hydrocephalus by removal of the choroid plexus, it would be necessary to remove all of the choroid plexuses because there is no absorption in the ventricles; but in communicating hydrocephalus, there is about one-fifth of the normal absorption. It would not therefore be necessary to remove the entire amount of choroid plexus but to reduce its volume until the amount remaining would not produce cerebrospinal fluid faster than it could be absorbed. In other words, it would be necessary to remove roughly four-fifths of the total amount of choroid plexus to reduce the fluid formation to the 8 to 10 per cent. absorption (one-fifth of the normal) which occurs in communicating hydrocephalus.

It is not feasible to remove the choroid plexus from the third ventricle and very difficult to extirpate flocculi in the fourth ventricle. Roughly the choroid plexuses of the combined third and fourth ventricles is about one-fifth of the total amount in all the ventricles. The plexus is in the two lateral ventricles therefore comprise about four-fifths of the total. It is relatively easy to remove the choroid plexus from both lateral ventricles. A bilateral extirpation from the lateral ventricles should, therefore, according to our present conceptions, reduce the formation of cerebrospinal fluid to a point where it can be absorbed by the restricted patent area of the subarachnoid space.

The problem, however, is not purely one of mathematics. Casually, one would expect that the removal of 75 per cent. of the productive structure would reduce the formation to meet absorption which is 20 to 25 per cent.

of the normal, but it should do even more, for in hydrocephalus fluid is formed at a greatly reduced rate because of the changed intracranial pressure from fluid accumulation. It has been demonstrated that the formation of cerebrospinal fluid is to a large extent at least mechanical. This can easily be demonstrated by inducing venous congestion by compression of the veins of the neck. We know that fluid forms at a greatly lessened rate in hydrocephalus due to the increased intracranial pressure, because if it formed at the normal rate (which we know) the head would grow at a tremendous speed. Although nature can modify the rate of fluid production she is unable to reduce its formation to the level at which it can be absorbed. The removal of the choroid plexus from both lateral ventricles should be more than is necessary, but it seems preferable to remove too much with no consequent danger, than to run the risk of an insufficient removal, in which event a progressive destruction of the brain will inevitably result. Hydrocephalus even when developing at the slowest rate causes a rapid atrophy of the brain. It is of course obvious that if more than the necessary amount of choroid plexus is removed, extra- and intravascular pressure differences will produce sufficient fluid to maintain the necessary amount of fluid to fill the ventricles.

The Operation.[8]—The steps in the removal of the choroid plexus are clearly shown in the accompanying drawings by Miss Norris. A small circular bone flap is made over the parietal eminence (Fig. 6). The wound is made well posterior to the Rolandic area and in a salient part of the occipital lobe. After ligating numerous vessels on the cortex by circumvection, the cortex is bloodlessly incised and this incision carried into the ventricle. From the exposure which is over the junction of the body and descending horn of the lateral ventricle, the entire extent of the ventricle can be brought into view (Fig. 7). The opening in the brain is maintained by an open nasal dilator (Fig. 8), or when the ventricle is very large the brain wall must be elevated by a spatula which is inserted into the ventricle. It is necessary to remove all the cerebrospinal fluid in the ventricle to get a view of the choroid plexus; the brownish-red flocculent choroid plexus can then be easily followed from the foramen of Monro to the tip of the descending horn.

The choroid plexus is picked up in forceps at the foramen of Monro (Fig. 8), and the vessels ligated by a silver clip. A pledget of moist cotton is inserted into the foramen of Monro to prevent blood gravitating into the third ventricle. The plexus is then transected and gently stripped backward from its narrow attachment to the floor of the body of the ventricle. When the glomus is reached the stripping from the body of the ventricle is stopped and the choroid plexus picked up at the tip of the descending horn (Fig. 9). This part of the choroid plexus is also stripped backward to the glomus; the remaining attachment of the glomus is then liberated and the entire choroid

[8] I am greatly indebted to Professor Halsted for many suggestions in the development of this operative procedure, as well as in the experimental work upon which the operation is founded.

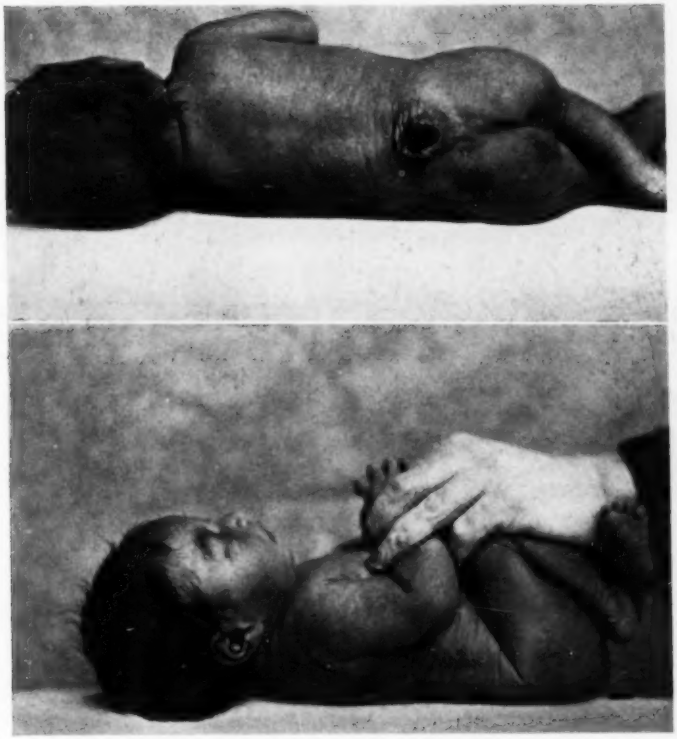

FIG. 1.—Views of case of early communicating hydrocephalus with lumbar meningocele. The meningocele was removed and a bilateral extirpation of the choroid plexus of the lateral ventricles was performed in the first three weeks of life. The head is not enlarged. A most unusual diagnosis of hydrocephalus was made at birth by Dr. J. Whitridge Williams, because of the wide fontanelles and the meningocele.

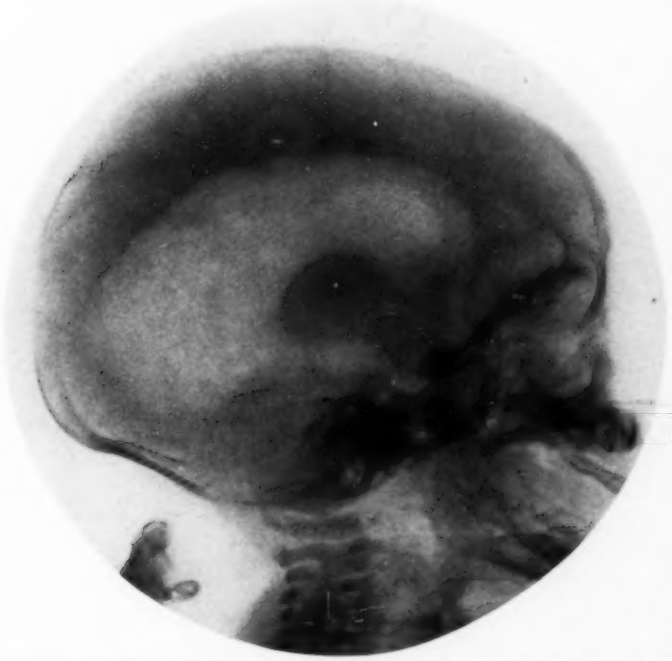

FIG. 2.—A ventriculogram of the case of communicating hydrocephalus shown in Fig. 1. A well-advanced hydrocephalus can exist even when the size of the head is normal.

FIG. 3.—Same child as Fig. 1, eight months after the bilateral operation. The scar can be seen in the back of the head. The head is still of normal size. The size of the fontanelles is much reduced. The child is apparently perfectly well.

FIG. 4.—Case of advanced communicating hydrocephalus. Beyond this stage the patient seldom survives any operative procedure. This patient died three weeks following the removal of the choroid plexus of one lateral ventricle.

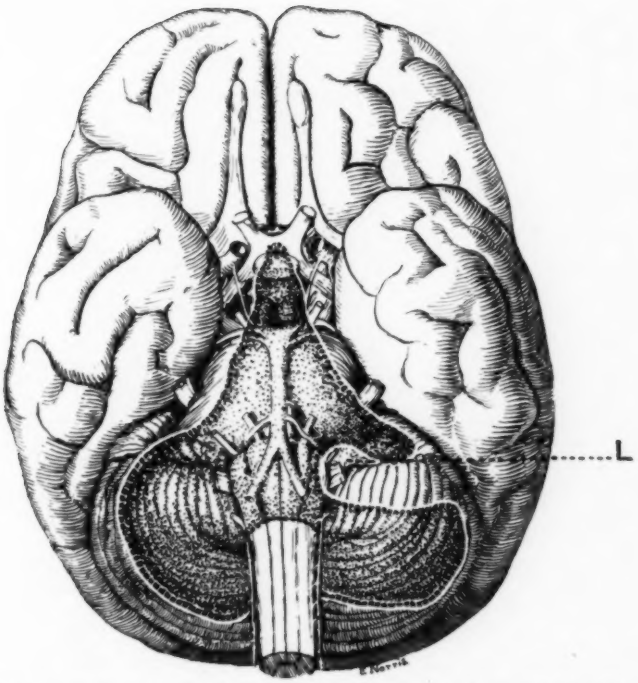

FIG. 5.—Diagram to show the distribution of adhesions over the base of the brain in a case of communicating hydrocephalus. All the cisternæ are obliterated. Communication between the ventricles and subarachnoid space is restricted to one foramen of Luschka (L). The other foramen of Luschka and the foramen of Magendie are sealed by the adhesions. Ablation of the cisternæ prevents cerebrospinal fluid reaching the cerebral subarachnoid space.

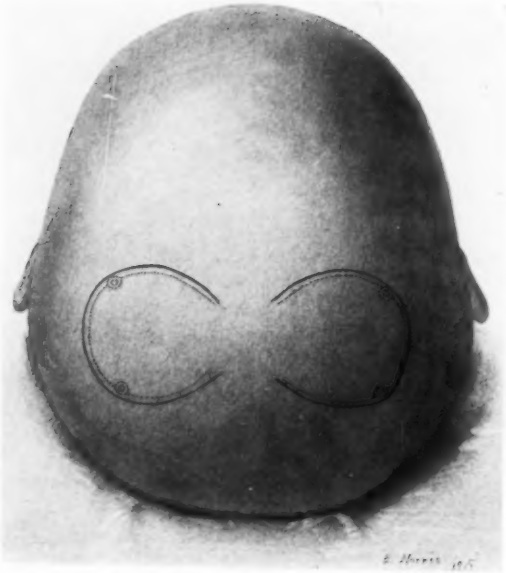

FIG. 6.—Shows location, shape and size of incision in scalp and bone for bilateral extirpation of choroid plexus of lateral ventricles. The solid line marks the skin incision, the dotted line the bone flap which is broken and reflected at the base.

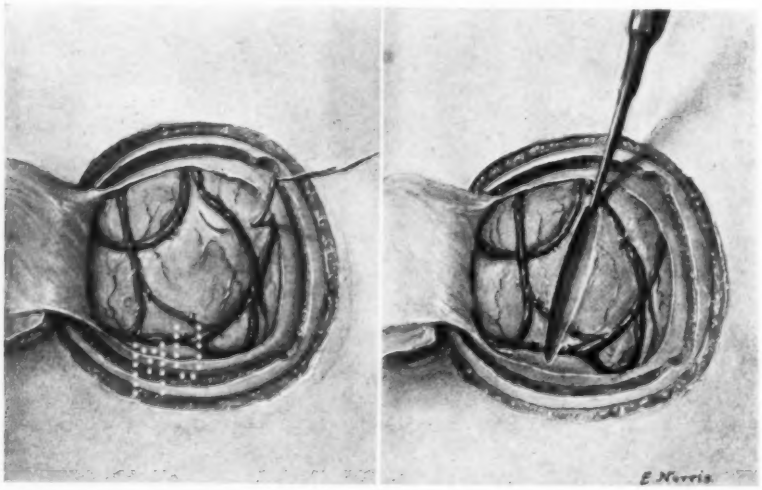

FIG. 7.—Skin, bone and dura are reflected. The cerebral vessels are doubly ligated with fine silk by circumvection. Between these two rows of sutures the cortex is incised with the scalpel and this incision carried through to the ventricle.

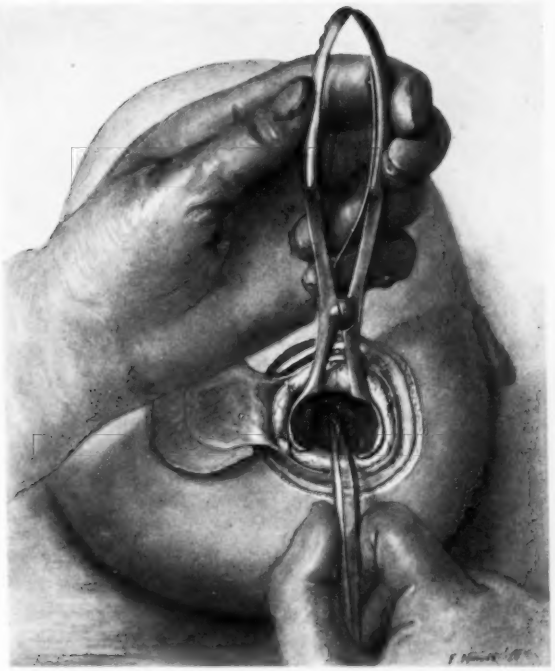

FIG. 8.—The opening in the cortex is maintained by a nasal dilator. Through this opening the choroid plexus is removed in the manner shown in Fig. 9. The fluid has been entirely removed from the ventricle to permit exposure of the choroid plexus.

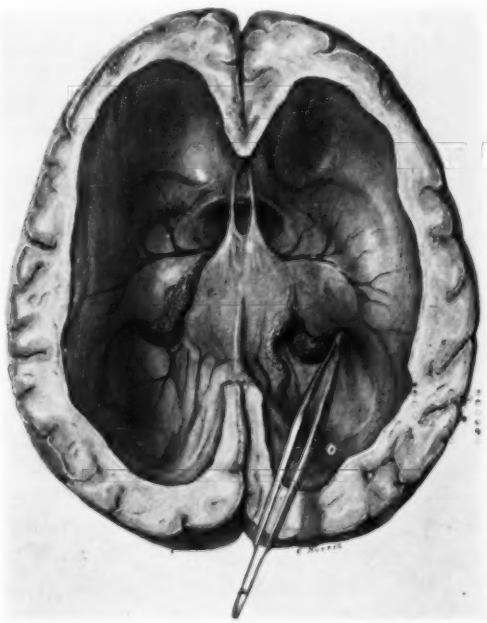

FIG. 9.—Coronal section through hydrocephalic brain, showing method of stripping choroid plexus from its attachment to the floor of the ventricle. The right plexus has been stripped from the foramen of Monro to the end of the glomus and at the tip of the descending horn is shown grasped by the forceps in the process of being stripped to the glomus. The entire plexus is then lifted from its bed.

plexus removed in toto. Bleeding from the denuded area of velum interpositum is slight and easily controlled by moist saline cotton pledgets. Special care must be taken to leave no bleeding points.

The collapse of the brain following evacuation of the ventricular fluid causes a remarkable infolding of the cerebral walls, the extent depending of course upon the size of the ventricle and thickness of the cortex. In advanced cases a tremendous cavity results, which is filled before closure with salt solution to restore the collapsed cerebral wall as nearly as possible to its natural convexity.

A remarkable exposure is obtained during the operation in the ventricle. One can see the third and opposite lateral ventricle and the septum lucidum which is frequently perforated in many places owing to pressure atrophy.

The opening in the cortex is closed with a series of interrupted fine silk sutures which are held by the delicate pia arachnoid membrane. The dura and scalp are carefully closed also with silk, special care being taken to prevent any subsequent leak of cerebrospinal fluid.

Result.—I have extirpated the choroid plexus in four cases of hydrocephalus from Professor Halsted's Clinic. All of these have survived the operation, although three died two to four weeks after the operation. One patient has survived a bilateral choroid plexectomy ten months, and shows no evidence that the disease is advancing. During and following the operation the reaction seems to vary directly with the grade of the disease. If the ventricle is small the operation will be well tolerated even by a very young baby. When the ventricles are large and the cortex is greatly thinned and marked enlargement of the head has resulted, a very severe reaction occurs during the operation and the convalescence is very slow. In the highest grades of hydrocephalus death will follow almost immediately upon release of the fluid, or a very severe reaction will result at once and death will quickly follow. In the advanced cases we can hold out very little encouragement from operative procedures of any kind which will necessitate release of fluid and consequent collapse of the brain. There is, however, little object in attempting a cure of the disease in this advanced stage because the child would be left a hopeless imbecile. In the three cases of this series which subsequently died the hydrocephalus was of the extreme grade. In each of these there was an immediate operative collapse, beginning with pallor of the face and body, rapid feeble pulse which quickly becomes imperceptible, cold clammy perspiration, rapid, shallow and irregular respirations—in other words, typical shock. These changes invariably begin with evacuation of ventricular fluid, and are undoubtedly due to differences of pressure which affect all the blood-vessels and directly or indirectly the centres in the medulla. Following escape of the fluid the thin brain walls collapse like a wet cloth. In addition to the differences in intra- and extravascular pressures, the mechanical kinking of the large vascular trunks by angulation of the infolding brain must have a pronounced effect upon the circulation.

In each instance there was a gradual recovery, and death came from two to four weeks later. In two cases death was no doubt due to gradually progressive acute intracranial pressure which we now would be able to recognize and probably alleviate. In a third case the cause of death is still uncertain, though undoubtedly a result of the operation. The temperature rose to 108° four hours after operation; remained around 102° to 106° for two weeks. Though conscious, the ability to swallow was lost and not regained. At autopsy no cause for death could be observed.

In the fourth case, which is still living and well and with no evidence of progress of the disease, no post-operative effects were observed, despite the fact that within three weeks of birth three operations were performed, one for the removal of a large myelomeningocele and two for the bilateral extirpation of the choroid plexus. Feeding was uninterrupted; the temperature at no time rose over 100°, and the rising curve of body weight was not even temporarily affected.

This case was kindly referred to me by Dr. J. Whitridge Williams, who made a most unusual diagnosis of hydrocephalus immediately following the child's delivery in his clinic. The head was not enlarged but the fontanelles were wider and a trifle fuller than normal (Fig. 1). The myelomeningocele also suggested the possibility of hydrocephalus. A ventriculogram[4] showed a well developed hydrocephalus with complete obliteration of the posterior horn of the lateral ventricle (Fig. 2). In no other way could this very early tentative diagnosis have been substantiated.

Unfortunately in this case our diagnosis of communicating hydrocephalus has been to a large extent conjectural. The large myelomeningocele filling the lumbar region precluded successful lumbar puncture, so that we were unable to determine by the phenolsulphonephthalein test whether communication was present, or indirectly by quantitative absorption whether the hydrocephalus was of the communicating type.

The absorption of phenolsulphonephthalein following injection into the ventricle was 2 per cent., which is a little higher than in obstructive hydrocephalus and about what obtains in communicating hydrocephalus. This is not considered conclusive by any means, as the difference between the ventricular absorption in the two types of hydrocephalus is not great enough to be a decisive test. The meningocele is by no means evidence in favor of a communicating and against an obstructive hydrocephalus.

Wherever possible both choroid plexuses should certainly be removed. This requires two operations, the length of time intervening depending upon the reaction following the first operation. Only one of our cases has had a bilateral extirpation. It is doubtful whether the removal of the choroid plexus of one ventricle would produce more than a retardation of the disease, which, of course, would be of no ultimate benefit.

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[4] Dandy, W. E.: Ventriculography Following the Injection of Air Into the Cerebral Ventricles. Ann. Surg., July, 1918.

Even though one case is apparently well ten months after a bilateral extirpation (Fig. 3), no conclusions are justifiable on the basis of a single case or in such a short period of time. The operation is presented without claims of cure, but because of its apparently sound scientific foundation it is hoped and expected that cures will result.

CONCLUSIONS

1. Any treatment of hydrocephalus must be based upon the etiology and pathology of this disease.

2. Communicating hydrocephalus is caused by an obstruction in the subarachnoid space causing diminished absorption of cerebrospinal fluid.

3. This obstruction is probably nearly always due to adhesions following meningitis.

4. These adhesions close the cisternæ through which all cerebrospinal fluid is distributed to the subarachnoid space over the cerebral hemispheres.

5. Absorption is reduced to one-fourth or one-fifth of the normal, roughly corresponding to the volume of subarachnoid space which contains cerebrospinal fluid.

6. It is at present impossible to reëstablish the cisternæ by surgical means.

7. By experiments it has been demonstrated that cerebrospinal fluid forms from the choroid plexus.

8. An operation is presented for the cure of this type of hydrocephalus by removal of the choroid plexus of both lateral ventricles. This removes, roughly, four-fifths of the total amount of fluid-forming structures. It is hoped the cerebrospinal fluid which forms from the choroid plexus can be absorbed in the small amount of subarachnoid space which remains in the third and fourth ventricles.

9. Choroid plexectomy is of value only in communicating hydrocephalus. Any treatment therefore presupposes an accurate diagnosis of the type of hydrocephalus. This is best made by the phenolsulphonephthalein test.

10. The operation has been performed on four cases. One case is alive and apparently well ten months after the operation.

11. Sufficient time has not elapsed to speculate on the practical results of the operation.

12. The operation can be safely performed in moderately advanced cases of hydrocephalus.

RIB CARTILAGE TRANSPLANT FOR SADDLE-BACK NOSE*

By Penn G. Skillern, Jr, M.D.
of Philadelphia

Case I.—Male, white, aged twenty, clerk, who fifteen years previous to admission suffered fracture of the nasal bones. Examination reveals depressed bridge of nose (Fig. 1). The patient is blind in the left eye, due to lues. He received intensive arsenobenzol treatment.

Operation was performed under local anæsthesia (novocaine ½ per cent.). Incision made over seventh right costal cartilage close to sternum; tissues retracted; section of cartilage, 2 inches long by ¼ inch broad by ⅛ inch thick, removed and placed in normal saline; field sprayed with dichloramine-T; hæmostasis effected; wound closed with interrupted sutures of silkworm-gut; one piece of rubber dam placed in outer angle of wound for drainage; dry gauze dressing.

Novocaine ½ per cent. subcutaneous infiltration made in midline of nose from glabella to tip. Vertical incision ½ inch long in mid-nasal furrow: tissues retracted; periosteum of frontal bone exposed. With Mayo curved dissecting scissors a subcutaneous tunnel was made from glabella to tip: scissors withdrawn, tunnel packed with dry gauze. Horizontal incision ½ inch in length made in periosteum of frontal bone. Cartilage transplant removed from saline solution, gutter made in deep surface. Transplant with perichondrium uppermost inserted into subcutaneous nose tunnel, after removing gauze packing from latter and mopping latter with dichloramine-T: upper flap of periosteum of frontal bone raised with blunt hook, upper end of transplant inserted beneath same. Incision closed with one suture of silkworm-gut. No dressing was applied (Fig. 2).

Case II.—Male, white, aged twenty-four, single, pugilist, who was admitted to Polyclinic Hospital (Case Record No. 324-9) on December 11, 1917. The patient as a pugilist had received numerous blows upon the nose, resulting several times in fracture. In consequence the bridge of the nose is depressed, this forming a variety of saddle nose (Fig. 3). The patient states that at one time after receiving an injury he blew his nose and the eyelids filled with air. Physical examination reveals a naturally broad nose, the bridge of which is depressed, so that the anterior nares face somewhat forward. The operation was essentially the same as in the previous case, except that a transversely curved incision was made between eyebrows and with convexity downward. The result is shown in Fig. 4, the patient's friends agreeing that his nose had been considerably improved by the operation.

According to the experiments of Dr. John Staige Davis, of Baltimore, cartilage transplants are practically never absorbed, while bone transplants unless accurately contacted with bone and periosteum lead a precarious

* Presented before the Philadelphia Academy of Surgery, May 2, 1918.

FIG. 1.—Saddle nose before operation; note short distance between eyelashes and surface of bridge of nose.

FIG. 2.—Nose after operation: note great increase in distance between eyelashes and surface of bridge of nose.

FIG. 3.—Case II. Saddle nose before operation.

FIG. 4.—Case II. Nose after operation.

FIG. 5.—Author's test for determining approximate size and shape of graft to be inserted.

existence, and are ultimately more or less absorbed. Cartilage has the further advantages of being easily obtained and easily pared with a knife to the desired pattern, while by its elasticity the intervening portion between the ends, which are supported by the frontal bone above and the septal cartilage below, takes up the slack in the skin, thus filling the latter to the required level and without undue tension; the graft itself is not subject to fracture in the event of a blow upon the nose, as would obtain were a bone graft employed. Regarding the use of dichloramine-T, the subcutaneous nose tunnel was first packed with gauze saturated with this substance, thus sterilizing the space and taking up whatever oozing there might be, just as when preparing a pocket in the scrotum for the reception of an undescended testicle. One silkworm-gut suture was used to close the wound, which was left exposed to the air. In the first patient there had been some transitory œdema over the forehead after operation, but in the second patient postoperative œdema did not take place. As regards the choice between the supranasal and the intranasal routes for insertion of the graft, the objection to the latter would be the danger of infection spreading to the graft field from the nasal cavity, especially bearing in mind the advice of the late Dr. John B. Murphy—namely, when preparing the pocket for reception of the graft to take great pains to avoid opening into the nasal cavity with the dissecting gynesic scissors, in order to avoid the ever present danger of infection from this source.

The frontal bone is nourished and reproduced from the dura, the periosteum covering it being devoid of osteogenetic properties: for this reason burial of the upper end of the graft beneath the periosteum of the frontal bone is not done with any expectation of getting nourishment or reproduction of bone from that source, but it is done with the idea of more firmly securing the graft *in situ*. Recent experiments show that cartilage when transplanted establishes nutritional relationship with its host just as surely as bone does.

In Figure 5 is shown a test devised by the author for determining the approximate size and shape of the graft to be inserted. With thumb and index finger applied to base of nose at its middle draw the skin forward: the skin over the bridge will now resume its normal conformation, and a space will be found between the skin and the bridge, representing the amount of height-loss of the bridge and therefore the thickness of the graft; the anterior nares will drop downward to their normal site.

ARTERIOMESENTERIC ILEUS OF DUODENOJEJUNAL FLEXURE

By William H. Fisher, M.D.

of Toledo, Ohio

surgeon, st. vincent's hospital

In looking over the literature of this subject, one is impressed with the confusion that exists as to its causation; the misnomers that are applied for this condition; and the various inefficient measures advocated for its relief.

It is the purpose of this paper to present various facts and salient points that will aid in clarifying this subject and establish it as a true surgical entity with the proper method of treatment.

Literature abounds with reports of cases with the title of gastromesenteric ileus, acute dilatation of stomach, acute gastroduodenal dilatation, giant duodenum, acute and chronic duodenal dilatation. All these are the effects of the condition named in the title of this paper and belong to and are a part of the symptom complex, and as such should be discarded, having no claim *per se* in the nomenclature of surgical entities. For convenience of elucidation, let us divide this subject into three distinct clinical types of obstruction: (1) Organic ileus; (2) acute arteriomesenteric ileus; (3) chronic arteriomesenteric ileus.

Before proceeding with the etiology, the anatomical arrangement of this flexure should be considered.

This first true flexure of the viscera is at the end of the fourth portion of the duodenum. The ascending portion passes from the right upward, over the fourth and third lumbar to the second lumbar vertebra, then continues as the jejunum: the duodenojejunal flexure being situated at the left side of the body of second lumbar vertebra. The longitudinal musculature of the ascending portion is reinforced by a broad flat band arising close to the abdominal aorta from the crus of the diaphragm and known as the musculi suspensorius duodeni (muscle of Treitz); it serves to maintain the duodenojejunal flexure in position.

Albrecht pointed out that this segment of the duodenum has under normal conditions, not a round contour but a distinctly flattened circumference. Codman presented casts of a number of duodenums showing definite evidence of compression.

Etiology.—Braun, Seidel, and Kayser advocate the view "that dilatation is primary in stomach; that duodenal dilatation, if present, is secondary and brought about by kinking at the mesentery. The latter is due to pressure of stomach on the mesenteric vessels and traction of the mesentery of the small intestines. The primary acute dilatation of the stomach is brought about by the action of some toxins on its nervous mechanisms."

Bloodgood reports in two cases "that relief of tension on the mesentery

and jejunum, pressure on the duodenum and pushing up the dilated stomach did not relieve the obstruction nor allow the duodenal contents to flow into jejunum."

Moore reports two cases of gastromesenteric ileus. At autopsy the pressure of the stomach on the last portion of the duodenum was relieved by turning up the transverse colon over the chest wall. The jejunum remained collapsed and remained so even after moderate pressure had been made simultaneously on the anterior and posterior surfaces of the stomach. After the stretch of the root of the mesentery was relieved, stomach lying in position, the jejunum continued to remain collapsed, but when slight pressure was made upon the anterior surface of the stomach, the jejunum would fill rapidly. The contents of the jejunum were stripped into the duodenum and held there by traction on the root of the mesentery. When the stomach was put in place and the stretch of the mesentery relieved, the jejunum began to fill up, but would cease to fill when slight traction was made upon the mesentery.

More of these cases are reported, but these are illustrative, and an analysis reveals dissimilarity in manipulative results and they are readily explained by a further study into causation.

It is not the purpose of this paper to enter into the etiology of the first clinical type or organic ileus of duodenojejunal flexure. The factors that produce it and the pathology are self-evident. But what of the second clinical type, that group of cases where dilatation extends to the flexure, in which there is no evidence of pathological conditions to explain the obstruction other than the normal anatomical factors?

Jordan describes a vivid fluoroscopic picture of a dilated duodenum. The duodenum was half as long again and more than double the width of a normal duodenum. For seven or eight minutes the duodenum was observed undergoing rigorous writhing contractions in a vain endeavor to force its contents through the kink at the duodenojejunal junction. After seven or eight minutes a very powerful contraction of the duodenum forced a large mass of bismuth emulsion through suddenly into the jejunum, and the bismuth forthwith began to arrive rapidly through the coils of the small intestine.

This case demonstrates the duodenal musculature had sufficient propulsive force to overcome the partial occlusion.

There is a greatly altered physiological relation and increase of the secretions of stomach, liver, and duodenum, due directly to the effects of anæsthesia, especially true in infective cases. The inhibiting influence of narcotics given to relax patient is a contributing factor in the production of gastroduodenal stasis, dilatation with tension ensues and with tension regurgitation. The greater the dilatation the greater the tension, and the greater the tension the less propulsive force of duodenum and the greater the obstruction at flexure. The duodenojejunal flexure ceases to be a normal physiological one and becomes an occlusion, produced by innervation of musculature with duodenal tension.

Different subjects have varying degrees of angulation of the flexure; this angulation may assume a partial occlusion or be accentuated by certain contributing factors, as compression by mesentery or traction in visceroptosis. The severity of the occlusion then may be gauged by the degree of angulation, or the duodenal tension, or the mesenteric compression, or the visceroptotic traction, acting singly or conjointly.

Thus we may readily account for the controversial reports of cases that, lifting the root of the mesentery with the stomach and colon will allow the duodenal contents to pass into the jejunum; while in other cases, even pressure on duodenum with above manipulations fails to empty duodenum.

This principle in physics is readily demonstrated by suspending a rubber tube in the fixed anatomic position as the duodenum and filling it with water. There will be distention of tube below the point corresponding to duodenojejunal flexure. The more the distention the greater the occlusion. The atonic duodenum, with its diminished propulsive power, is practically an inert tube, and with its increase in dilatation and consequent tension changes a normal physiological flexure into an occlusion.

The duodenum has two other flexures which are not pathologically significant, except congenitally, and are not subject to acute angulation. The result is regurgitation of duodenal contents into stomach. Many of these cases recover under proper medical treatment and recovery is due to the rehabilitation of splanchnics and duodenal musculature.

I am convinced that the foregoing is a true explanation of the causation of intestinal ileus at duodenojejunal flexure in the absence of any appreciable pathological state.

Etiology of the Third Clinical Type.—In the consideration of the etiology of the third clinical type, Conner in discussing "Dilated Duodenum" aptly says: "The mechanical factors involved are still, it seems to me, very obscure. There can be no doubt that the obstruction by the root of the mesentery and by the superior mesenteric artery is a real one; but when one attempts to explain what the conditions are that bring about such an obstruction, there are many difficulties. Certain things are obviously necessary; if the mesentery is to pinch the duodenum, it must be tight and there must be traction in a certain direction. Those conditions are fulfilled by the small intestine being in the true pelvis and being empty; it cannot be in the true pelvis unless it is empty. Apparently the mesentery must be neither too short nor too long in order to constrict the duodenum."

Compression then by the root of the mesentery or traction, either singly or conjointly, are the main causes of occlusion. But the essential contributing factor is the anatomical feature of the flexure. The fixed duodenum, with its varying degrees of angulation, explains succinctly many of the hitherto obscure mechanical factors involved in the production of ileus at duodenojejunal flexure.

Treatment.—M. Wilms, in discussing high intestinal obstruction, says: "The time in which duodenojejunal obstruction causes death has only in the

rarest instances been prolonged. Death usually subvenes with extreme rapidity. All patients succumb to toxic absorption."

Turck has experimentally shown that in such atonic dilatation, "Colloid material can penetrate unchanged into the walls of the intestine and can pass along the submucous tissue cephalad. Reaching the upper intestinal tract they are split up by the action of powerful enzymes in the wall and toxic effects are produced."

When the above condition subvenes in an occluded duodenum toxæmia rapidly obtains with fatal results. To forestall this state and properly drain the duodenum is the indication of medical and surgical treatment.

Murphy in writing of congenital occlusion of small intestines advocated: "When the occlusion is below the papilla a duodenojejunolateralis can readily be performed. The same operation is applicable in occlusion at the duodenojejunal flexure."

Bloodgood, reporting three cases of acute dilatation of stomach, says: "Theoretically, also a second anastomosis between the duodenum and jejunum would have to be performed to drain both the duodenum and stomach. Future experience may demonstrate in cases of this character a simple duodenojejunostomy is indicated." And further in same article: "If the dilatation involves the duodenum I would advocate jejunostomy with passage of tube into duodenum or duodenojejunostomy."

Downes reports a case of giant duodenum in a child of four and a half years: "Posterior gastroenterostomy was performed, reoperated one month later and finally recovered." The author believes that duodenojejunostomy best meets the indications and should have been adopted in his own case.

Maury, in a series of animal experiments to determine the cause of death in high intestinal obstruction, among other facts deduces this important practical observation: "It was further determined that the effect of enteroanastomosis between the jejunum just distal to the gastro-enterostomy and the closed duodenal loop just aboral to the entrance of the bile-ducts gave a favorable result."

In other words, drainage of the obstructed loop is effectual in preventing lethal results.

Treatment First Clinical Type.—The treatment of the first clinical type is entirely surgical and the operative technic dependent upon the cause. Where possible, growths should be excised; strangulated hernia in duodenojejunal fossa should be relieved; effects of infective peritonitis corrected; enteroliths removed, etc. When not possible to remove cause and relieve the occlusion, the duodenum should be drained, either to prolong life or in expectation of permanent recovery, and duodenojejunostomy is indicated. The following case report of this clinical type is of interest:

History of Case.—Joseph R., American, age twenty-one years. Occupation, farmer. Family history negative. Personal history elicited nothing except stomach disturbances from childhood. Burning sensation in epigastrium associated with exercise. Bananas only food product

that disagreed with him, producing indigestion with distention. Always a tendency to constipation and, when not relieved, vomiting associated with it. Never any gastric pain in early course of disease. A very significant and pathognomonic symptom developed last April. On assuming the upright position, after working in a stooping posture, he was taken with an epigastric pain radiating to back and right of abdomen. Pain was continuous, more or less severe, and compelled him to cease work. Always more severe on regaining upright position.

Condition Found at Operation.—Right rectus incision June 19, 1918, revealed a dilated duodenum. The duodenojejunal fossa was entirely obliterated by dilated duodenum and surrounding retractive jejunal peritonitis. The jejunum instead of passing to left and downward was retracted *upward* and to *right,* firmly adherent to abdominal vessels and completely flattened against spine. Its course then was downward and to the right.

Operative Technic.—The surgical consideration was to restore the bowel to its natural position. After careful dissection the bowel was freed from its attachments, the mesentery unrolled, allowing the bowel to pass to the left. The raw surfaces were closed over and a retaining suture was placed on the jejunum to prevent further rotation to the right. The patient was elevated in bed and was kept on left side for three days. Convalescence uninterrupted. Discharged from hospital two weeks later. The keynote of success in the operative technic was the careful dissection to relieve the double angulation and in the proper juxtaposition of peritoneum over vessels, thereby avoiding a future retraction of bowel. Up to the present time he reports himself well.

Treatment of Second Type.—The treatment of the second clinical type is either medical or surgical. It has been customary for many years in my surgical service to have lavage of stomach performed immediately following operation on all septic cases, and I am more convinced of its value as a prophylactic measure in preventing gastroduodenal innervation. If after thirty-six to forty-eight hours, despite medical treatment, the tension of the duodenum has not been relieved, epigastric pain, vomiting and gulping, coprostasis, and meteorismus continue with evident dilatation of stomach and duodenum, immediate surgical intervention is indicated. In urgent toxic cases, to drain the duodenum with the least possible disturbance is the surgical technic of choice. Under local anæsthesia a jejunostomy should be performed and rubber tubing passed through flexure into duodenum. Through this tube the duodenum should be constantly irrigated. In less toxic cases a duodenojejunostomy should be performed.

Treatment of Third Clinical Type.—For the same cogent reasons that have led me to advocate duodenojejunostomy, representing as it does anatomico-physiological, biological, and surgical principles, so also must I discountenance the medical treatment of this clinical type.

The medical treatment advocated, is rest in bed over a long period of time; in assuming various postural positions to relieve epigastric distress, and with forced feeding in hope of a deposition of fat in mesentery to prevent its undue compression or traction. As I have

previously stated, this clinical type of cases is produced by three factors that influence the flexure, viz., anatomical features, compression and traction, compression by mesentery and traction of visceroptosis. The effects produced are identical in either factor, *i.e.*, atony of duodenum with dilatation and varying degrees of toxæmia.

From the preceding, duodenojejunostomy is positively indicated. Slitting the mesentery is unsurgical; gastro-enterostomy has not one surgical principle to commend it and both should be discountenanced. Reports of favorable cases have been made of resection of one-half colon with ileocolostomy for the relief of occlusion at flexure. These cases are of the traction type of visceroptosis. Why such a formidable operation in these cases? Unless directly drained, time is an important element in the restoration of the atonic duodenum. In the interim, contributing factors may intervene and produce lethal effects of this "reservoir of toxicity."

The occlusion of duodenum in lordosis of lumbar spine is due to both compression and traction. A formidable resection is certainly contra-indicated here. A duodenojejunostomy is the operation of choice and if this operative procedure is tenable in such a case, why not more so in less grave cases?

CONCLUSIONS

1. Arteriomesenteric ileus at duodenojejunal flexure is a true surgical entity.

2. That the anatomical features are predisposing factors to occlusion.

3. That it has three clinical types: (1) Organic arteriomesenteric ileus; (2) acute arteriomesenteric ileus; (3) chronic arteriomesenteric ileus.

4. That each type has its specific causative factors: (*a*) Occlusion due to new-growths, infective peritonitis, strangulated hernia, cicatricial contraction, enteroliths, etc. (*b*) Splanchnic innervation with duodenal tension and resulting loss of propulsive power. (*c*) Compression by mesentery and its vessels or traction from visceroptosis acting either separately or jointly.

5. That surgical intervention is indicated and with the above noted exceptions duodenojejunostomy is the operation of choice.

PRIMARY RETROGRADE INTUSSUSCEPTION OF THE SIGMOID ASSOCIATED WITH TUMOR

By Donald C. Balfour, M.D.
of Rochester, Minnesota

We have recently observed, in the Clinic, a most striking example of a retrograde intussusception occurring in association with a sigmoidal tumor.

Power, who made such valuable contributions to the subject of intussusception, states that about 5 per cent. are of the retrograde type. However, it is quite obvious from such records as are available of these reverse types of intussusception that the case I present[1] is unique for the reasons that the condition was primary and had not developed as a complication to an intussusception of the usual variety, that it was not a terminal event in peritonitis or obstructive vomiting, and, finally, that the method of its recurrence could be observed at operation.

The patient (Case A203308), a male, aged forty-five years, presented himself for examination November 17, 1917. He had been in good health (the appendix had been removed twelve years previously) until October 1, 1917, when a diarrhœa began with from eight to ten watery stools a day, but without blood or mucus. The general physical examination was negative except for a slight abdominal tympany. The blood, stomach contents and urine, and the Wassermann test were negative, as were also repeated proctoscopic and stool examinations. The customary therapeutic measures were suggested, but on March 4, 1918, the patient returned to the clinic reporting that the diarrhœa, still without blood or mucus, had persisted. He had been disturbed by considerable flatulence and borborygmus, and by occasional moderately severe abdominal cramps—the history of an intermittent obstruction. The general examination again was negative, but X-ray of the colon showed a filling defect high in the sigmoid, a finding which remained after antispasmodics had been administered. March 30, 1918, the patient was sent to the hospital, and an exploration (W. J. Mayo) through a left rectus incision revealed a mass in the pelvic sigmoid. There was marked obstruction from the tumor, the sigmoid above the growth showing great distention and muscular hypertrophy. On further examination of the tumor (which through the bowel felt like a bunch of angle worms), it was discovered that a retrograde intussusception had occurred (Fig. 1), and that the tumor and the section of bowel involved had been intussuscepted upward about three inches. Within a minute or two after the reduction of the intussusception (Fig. 2), which could readily be done by traction, a strong reverse peristalsis was manifest, and the tumor was again drawn upward by powerful antiperistaltic contractions of the proximal sigmoid, these

[1] Dr. V. C. Hunt has kindly assisted me in reviewing the literature of the subject.

FIG. 1.—Appearance of retrograde intussusception at operation.

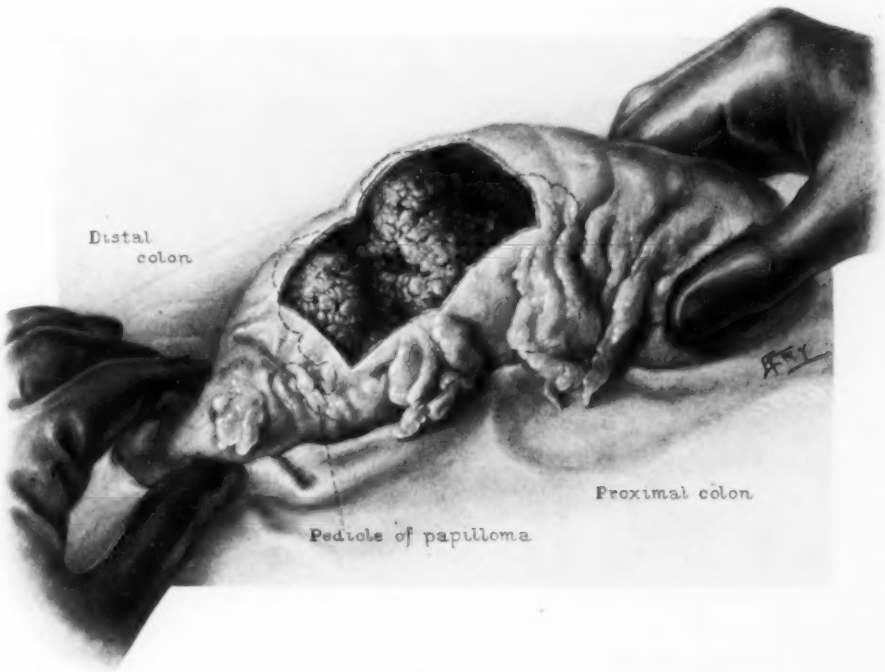

FIG. 2.—Intussusception reduced, portion of wall of sigmoid removed to show the tumor *in situ*.

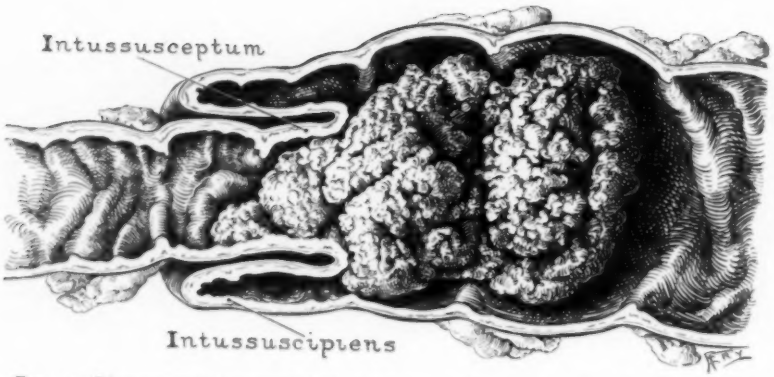

FIG. 3.—Diagrammatic representation of intussusception and tumor cut longitudinally.

contractions extending upward for a distance of from twelve to fourteen inches above the site of the tumor. The process of invagination, begun in this manner, continued until the portion of the bowel containing the tumor was drawn upward and completely engulfed by the proximal segment (Fig. 3). Ten inches of the sigmoid, including the growth, were resected. An end-to-end colonic anastomosis was done, and the suture line was drawn through the omentum (C. H. Mayo method). There were no enlarged glands, and exploration of the liver and peritoneal cavity was otherwise negative. The tumor itself proved to be a pedunculated malignant papilloma 10 by 15 cm. (Fig. 2). The abdomen was closed without drainage, but two small rubber tissue drains were inserted down to the peritoneum, and a tube was stitched in the rectum. The convalescence was uneventful.

The various theories as to the factors necessary to produce an ordinary intussusception may be applied in explaining retrograde intussusception in the colon. The first and most important fact is that, normally, muscular contractions in the large bowel are, at times, antiperistaltic. This is not true of the small intestine, consequently retrograde intussusception in the small bowel is almost always a terminal event, associated either with an ordinary intussusception or with the reverse peristalsis of obstruction. In the large bowel antiperistaltic waves have been demonstrated by Cannon, and he has shown that these waves may be and are produced by a tonus ring, although the conditions which establish the rings are not as yet determined. These antiperistaltic waves, which are the primary causative factor in retrograde intussusception in the colon, were, in our case, undoubtedly initiated by the tumor, the attachment of the base of the tumor causing a tonus ring just as such rings may be caused experimentally. Further, pedunculated tumors are the only variety in which an intussusception may occur; carcinomas, diverticulitis, and inflammatory tumors involve the intestinal wall to an extent sufficient to produce a rigidity which prevents invagination, and these tumors are not pedunculated.

The rarity, therefore, with which tumor formation in the sigmoid is associated with intussusception is because of the rarity of the type of tumor essential to such an occurrence. Given the factor of irritation, abnormally strong peristaltic waves are produced both above and below the site of the tumor. In the case under discussion not only did the tumor act as a point of irritation, but the polypoid mass, which was the size of a large orange, lay free in the colon, and as the powerful contractions of the hypertrophied musculature of the colon gripped the tumor they exerted enough pull on the base of the tumor to begin an invagination and a true retrograde intussusception. This invagination was aided, of course, by other factors, the most important of which were the difference in caliber of the bowel above and below the point of partial obstruction occasioned by the growth, the spastic condition in the lower segment, and, finally, a sufficiently long mesentery to give free mobility.

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A CONTRIBUTION TO THE STUDY OF MYOSITIS OSSIFICANS PROGRESSIVA

By Julius Rosenstirn, M.D.
of San Francisco
Surgeon to Mt. Zion Hospital

(From Mt. Zion Hospital, San Francisco, Calif.)

(continued from page 520)

Author's Case

L. B., newsboy, born 1896 in Rumania; normal confinement. Family history negative; both parents alive and well but very ignorant, and the history (previous to his coming under our observation), having been extracted from them under great difficulties, is not as complete as I should wish.

Two brothers (nine and twelve) and one sister (fifteen years) in good health. No T. B. or history of any other systemic disease in immediate family. Immediately after birth the midwife noticed a tumor in the right groin the size of a small egg. This tumor probably contained bony spicula; as the mother, who gave the details of the previous history, describes it as having felt to the touch as though filled with nails. His right leg was also shorter than the left. The attending physician advised a watchful waiting and to postpone surgical interference for one year.

At the end of that time the tumor had grown down the thigh to the leg, while the portion in the groin had grown to the size of a fist. He was operated on at the age of fourteen or fifteen months by a Rumanian surgeon who removed the tumor in the groin, leaving the remainder until the child had grown older and stronger. He stayed several months at the hospital. The parents, somewhat later, emigrated to the United States and at the age of five years patient was placed in the New York Orthopædic Hospital for a second operation.

An incision was made from the lower third of the thigh to middle of leg and a part of the tumor apparently successfully removed. The functional result was not improved, the leg remaining stiff, neither flexion nor extension being possible.

After three years patient again entered a hospital and remained there another eight months. It is not clear what was done at that time; parents say that a tumor in the upper part of the leg was excised. No further operation was done until he presented himself in my surgical Out-patient Department of the Mount Zion Hospital, under the care of my then first assistant, Dr. A. Newman, now Professor of Proctology C. P. & S., in the latter part of 1908. He complained of pain in the left upper arm and over his right tibia below the knee.

His status at that time was: A rather slender boy, good color.

Head and neck nothing abnormal. Left arm shows bony ridge in and under deltoid muscle, extending from below deltoid process and

along anterior outer surface of humerus to insertion of deltoid tendon. Humerus thickened; patient cannot raise his arm on account of pain when attempt is made to bring deltoid into action. Both thumbs show very short and broad last phalanx.

In the right groin a scar and another extending over inner part of right thigh, both from former operations. Exostotic growths spring from crest of right iliac bone and along the line of Poupart's ligament. There are small isolated isles of bony masses on the abdomen in the right iliac region, extending into right obliquus and rectus abdominis half way up to the umbilicus.

The entire adductores region of the right thigh, from their origin down to the lower third of the thigh, stonehard. The anterior part of thigh between lesser and greater trochanter also forms a bony mass; the femur is thickened; as the mass nears the knee it swings backward and entirely fills the popliteal space. Knee completely ankylotic. The mass then passes down along the inner side of the leg into the foot, running along the inner margin of the latter to the big toe. Microdactylia of both fourth toes.

Priessnitz compresses were ordered, to be changed every four to six hours. The pain disappeared soon after they had been regularly applied.

Entered hospital on August 31, 1909, with pain in left humerus and lower epiphysis of left ulna, and over right tibia below knee. Attack accompanied by fever. X-ray of left arm and hand shows thickening of upper two-thirds of humerus and periosteal thickening along lower part of ulna. In humerus just above epiphysis, oval wedge-shaped space giving impression of cavity. Tuberculin test negative. Under treatment with warm Burow compresses pain gradually disappeared but left thickening along ulna. Discharged October 2, 1909.

He was not seen again until the latter part of September, 1910, when he came into the clinic complaining of severe pains in the left upper arm. It was swollen, and painful to pressure and percussion all along the upper two-thirds of the humerus, and the skin appeared slightly œdematous. The pain had begun about a month before, but his great fear of a possible operation had kept him away from us until the pain had grown too violent to bear. Rest, ice and diluted Burow solution compresses were first tried, upon the urgent request of the boy, but gave no relief, and on October 2, I made an incision over the anterior and outer aspect of the humerus through the fibres of the deltoid, from the shoulder downward along the upper two-thirds of the bone. In the region of the insertion of the deltoid tendon the bone was bared, and on finding considerable roughness, an opening was made into the medullary cavity. A small quantity of pus was found in the medullary cavity (two cultures taken), together with considerable granulation tissue. The upper two-thirds of the cavity of the shaft were opened and curetted clean, until normal tissue below and above was found. The cavity was dried with the Hollander hot-air instrument and an equal part mixture of carbolic acid and tincture of iodine applied over entire exposed surface, after all bleeding

had been stopped. A paste of plaster of Paris, composed of sterilized plaster in 1 to 500 cyanide of mercury solution to fill the entire cavity is put in and allowed to harden. Periosteum and muscle are united with interrupted catgut, and skin with continuous silk sutures. Uneventful recovery. (Culture staphylococcus; no T. B.)

In the latter part of April, 1911, the patient came to us again complaining of considerable pain in the right iliac region during locomotion but not while at rest. Examination shows lower 5 cm. of right rectus muscle ossified. Mass very hard, a sharp-edged piece of bone raising skin tight.

May 13th: Incision 5 cm. long parallel to Poupart's ligament and about 1½ cm. internal from it near insertion of right rectus muscle. Muscle here completely ossified and a spur 2½ cm. long and 1½ cm. thick protruded down into the tendinous part, but entirely free from os pubis. It was removed, edges of muscle smoothed and wound closed. Discharged May 23, 1911.

A small piece from the lower edge put in trichloracetic acid for decalcification and microscopical examination.

On August 15th, 1911, returned to hospital. A large spur had formed, extending from the right anterior iliac spine to the edge of the obliquus externus of that side about 5 cm. long and 3 cm. wide, causing considerable pain. Incision over spur; blunt separation of adherent muscle and chiselling off of spur from anterior iliac spine. The rough bone edges smoothed off and hæmostasis with hot saline solution. Fascia united with chromic catgut; skin subcuticular. Uneventful healing; discharged August 30, 1911.

Returned in September, 1912, complaining about two closely neighboring ventral herniæ in the right iliac region where previously osseous plates had formed and which had been extirpated. The muscular fibres had atrophied and the ventral hernia developed. An attempt was made to bisect longitudinally a piece of the sound upper part of the right rectus abdominis muscle into an anterior and posterior half, and having tied off and resected the peritoneal sacs, turn the anterior flap down over the hernial apertures and fasten it to the lower tendinous part. The result was not very successful; part of the muscle-flap sloughed and the defect in the abdominal wall was only slightly improved.

About three weeks after operation the patient's face and both arms became œdematous and a marked hæmaturia appeared. The face and arms resumed their natural condition after two days, but the hæmaturia remained for several weeks. It finally cleared up without leaving any trace, nor has it ever returned since. (Late ether nephritis?)

A cystoscopic examination was stubbornly refused by the patient. Urine contained at first some granulated casts together with blood. After a few days casts disappeared. No pain or dysuria.

We did not see him again until May 26, 1914. As the status since then has remained about the same, I will give the one taken at that date.

Status.—Good complexion; red cheeks; looks like a boy of 15 years. Is underfed; weighs 113 pounds; rather infantile habitus; voice harsh and deep.

Head well-shaped and covered with an abundance of black hair. Face smooth, no sign of beard. Eyes clear, pupils equal, dilated, react to light and accommodation. Mouth: Mucosa normal, tongue clear; teeth carious, gums show signs of pyorrhœa alveolaris.

Pharynx inflamed, tonsils normal. Neck long, slender; no general glandular enlargement; thyroid gland not palpable; no visible pulsating blood-vessels.

Chest: Sternum in depressed groove, insertion of ribs on both sides forming convex arches to body and ensiform cartilage (ratite or ecarinate chest); entirely free from hair, which is also absent in the axillæ. Excursion on normal respiration is not marked. Breathing is mostly abdominal. On deep breathing, however, thoracic movements are extensive. Expansion is about 5 cm. Mobility on both sides equal.

Palpation: No muscular resistance; T. F. somewhat exaggerated on right side. Percussion and auscultation normal, slight bronchovesicular breathing below second and third interspaces. Slight lateral curvature.

The left arm shows a scar from the acromial process to the middle of the humerus, on its anterior and outer surface. The bone is thickened and rough and of irregular shape up to its middle, where it again assumes its normal size and contour. The change in form of the upper part consists of a nodulated groove 3 mm. deep on its anterior and outer surface. In the right iliac fossa, beginning about 3 cm. below level of anterior superior iliac spine, free osseous plates can be felt in the rectus and external oblique muscles. The skin is freely movable over them, but they are firmly imbedded in the muscle. Hardly any hair in pubic region.

From the right horizontal branch of the pubic bone an exostosis starts upward in the substance of the obliquus muscle for about 5 cm.

Its base is firmly attached to the os pubis; it is about one-half a ctm. wide and grows gradually wider toward the tip, where it is 2½ ctm. Near the tip a free osseous plate, at the edge of the right rectus, is movable and moves with respiration. This plate is about the size of a large melon seed. There are two smaller exostoses, one on each side of the large one, firmly connected with the pubic bone; the outer one, extending down to the large ossification of the thigh, connected with the femur; the inner one, near the symphysis, nearly joining the loose plate in the rectus above it. This exostosis is about 3 cm. by 1½ cm.

Between the outer and middle, also between the middle and inner exostosis, the muscle and skin are atrophied in two places and a ventral hernia protrudes through each of them, giving a marked visible and palpable impetus upon coughing.

The right thigh is ankylotic in hip joint and is flexed at an angle of about 35° and adducted. The knee is also ankylosed and in flexion at angle of about 25°.

The right leg shows ossification of the entire adductor and hamstring muscular groups; extensors and abductors not so well developed as on opposite side.

Circumference of middle of thigh—right, 30 cm.; left, 47.5 cm. Circumference just above patella—right, 28 cm.; left, 32.5 cm.

Nearly the entire mass of the inner and posterior muscles of the right leg is ossified; except the extensors, the peronei, the tibialis anticus, and the lower part of the soleus.

Circumference of calf—right, 24.5 cm.; left, 31.5 cm. Length of leg—right, 67 cm.; left, 80 cm.

Flexion of right foot is very limited, but movements of tendon Achilles are felt very distinctly. The foot is in adduction and its inner two-thirds ossified up to middle of metatarsal bones. The fourth toe of each foot is small and bent across third toe.

Patient complains of pain in the right lumbar and inguinal regions, particularly severe when standing erect. This pain commenced in a slight degree about eight months ago and gradually increased in intensity and duration, becoming lately so severe that patient was compelled to give up his work and apply to the hospital.

Examination: Shows small spur of bone in the right inguinal region about the middle of Poupart's ligament, about 1½ centimetres long and pointing towards horizontal branch of the pubic bone. Another sharp spur is on inner side of the femur, to the inside of crural canal.

Operation (May 28, 1914).—Incision over both spurs; the one in the abdomen can be easily removed by excision. Attenuated muscle and fascia are sewn together with catgut and skin with Michel clamps. The spur on the inside of the femur lies directly under the skin, and after incision it is removed with chisel; after hemorrhage has been stopped the skin is united with Michel clamps. Both wounds healed by first intention. Patient dismissed June 8th.

Returned to hospital October 25, 1915. Patient has been perfectly well ever since leaving hospital. About ten days ago he began to have pain over posterior surface of arm just above the wrist. This is a sharp sticking pain which comes on on movement of wrist or contraction of muscle of forearm. Greatest pain is when he flexes the wrist. Appetite good, bowels regular.

Examination reveals small highly sensitive exostosis, about the size of a small melon seed, over lower end of radius on dorsal surface; small hard nodule in *skin* above extensor tendon two inches above the wrist. Nodule is freely movable with skin and sensitive. Similar nodule over middle of left calf; underlying muscles and entire posterior part of left leg free from any other ossifications.

Heart and lungs normal.

A coagulation test of patient's blood was made, found normal; time 5 minutes.

Operation October 27.—For removal of exostosis on posterior surface of radius two inches above wrist joint. Esmarch's bandage applied to hand and arm, up beyond elbow. Local anæsthesia with novo-

caine. A longitudinal incision was made through skin, and subcutaneous fascia tendons of extensor muscles separated, deep fascia and periosteum incised and the latter levered from bone with elevator and pulled aside. The bared exostosis was then removed with bone forceps and base smoothed off with chisel. The small nodule with a small oval of surrounding skin was then removed under local anesthesia for microscopical examination, also that on left calf. Wound over wrist closed in two layers, deeper layer with interrupted catgut sutures; all skin wounds closed with Michel clamps. Dismissed October 31, 1915, after uneventful recovery.

Returned to hospital February 7, 1916. Complained of severe pain in right knee. Pain worse at night.

P. I.—About two weeks ago patient had a swelling on the medial side of the right great toe, which was very sore. At the same time the right knee on the lateral side became sore and a little swollen. The sore on toe did not break open but dried up and left a brownish scar. The knee continued to pain and kept the patient awake at night.

Examination shows swelling at outer part of right knee, occupying entire lateral surface and extending about 1 centimetre to the under surface into the popliteal region. Patient was confined to bed and a wet dressing applied to the knee.

February 13th: Pain in knee is less. Temperature varies from 37.2 to 38.4. Blood count: Leucocytes, 18,000; polys, 82 per cent. Patient refuses incision.

February 15. Cast applied to knee.

February 17: Pain worse. Cast removed and compress of Burow's solution applied.

February 18: Pain worse; temperature 37.9 to 38.4. W. B. C., 25,000; polys, 83 per cent.

February 19: Ice bag applied to knee.

February 20: Temperature rose to 39.9. Hot-water bag instead of ice bag. Pain somewhat relieved.

February 21: Patient taken to surgery. Two red inflamed areas were seen on the lateral aspect of the right knee. These were fluctuating and soft. Under local anesthesia the fluctuating areas were opened by incisions and a rubber tube inserted. A considerable quantity of yellow pus escaped.

February 22: Complains of pain on the inside of the knee. Highest temperature, 38.7. Similar nodules and swelling on inside of knee and indistinct fluctuation.

February 23: Abscess on inside of knee opened and drainage tube inserted. Highest temperature, 37.9.

February 24: Feeling much better; slept well; highest temperature, 37.3.

March 10, 1916: Two very small spiculæ of bone directly in skin; one on the right arm near insertion of deltoid muscle in outer groove, the other in popliteal fossa of right leg. These were extirpated, together with the skin containing them, for microscopical examination. Wounds sutured with silkworm gut.

March 15: Sutures removed from arm incision; union per primam.

March 16: Patient removed bandage from arm during night; wound broke open but quite clean. Edges approximated with adhesive.

March 17: Sutures removed from leg incision.

March 20: Hand and arm healing rapidly. Wound in leg clean and healing.

Discharged to come to clinic for treatment.

Small spurs of bone in the abdominal wall projecting with their sharp edges into the skin and giving considerable pain were removed on January 15, April 6, July 30 and December 30, 1917. The muscles and fasciæ in the places ossified by these pieces of bone have entirely disappeared and the lower surface of the bone is adherent to the peritoneum.

X-RAY FINDINGS. *March, 1918*—Hands: Both thumbs and second and third fingers show a diminutive last phalanx.

Abdomen, Pelvis and Right Thigh: The right side of the abdominal wall from the level of the crista ilii downward shows ossification in the muscular strata partly free, partly connecting with the pelvic bones. There is a scoliosis to the right in the lumbar vertebræ (compensatory). The pelvis is tilted from right to left. The right crista ossis ilii stands about 3 cm. higher than the left. The part of the os ilii forming the upper and posterior border of the acetabulum is much broadened and the entire frontal part of the right pelvis is obscured as through a thick veil by the ossification of the overlying soft parts so that the right foramen obturatorium seems obliterated in the picture. The neck of the femur stands in an angle of 160° to the shaft and the head is synostotically fixed in the acetabulum. The large trochanter is imperfectly developed, the smaller one relatively better. The shaft of the bone is less than one-half the thickness of the left femur. The entire anterior space ordinarily occupied by the adductor muscles is here changed to a solid mass of bone, which goes down to slightly above the internal condyle, where it merges with the contours of the femur and tibia in the formation of the completely synostosed knee-joint. The atrophic patella and its ossified ligament occupy their normal positions from the femoral attachment to the insertion at the tuberosity below the much-enlarged head of the tibia.

Right Leg: The outlines of the enlarged tibia can be traced in the picture. The soft parts in the popliteal fossa and all along the posterior part of the leg are nearly entirely ossified. The anterior part and the interosseous space are fairly free. The fibula is comparatively slender.

Right Foot: The foot shows an almost complete synostotic conglomerated mass of the ankle bones and calcaneus, together with the ossified muscles and tendons of the dorsum and part of the planta. There is a faint outline of the cuneiform bones at the base of the metatarsals and in the lateral view a similar one between the astragalus, the scaphoid and the first cuneiform. The metatarsal have retained their separate condition with their part of the interosseous muscles between them. The first metatarsal bone is very much enlarged in circumference through the ossification of its surrounding tissue; the fourth toe is microdactylic through a small metatarsal.

The left hip-joint shows a condition resembling coxa vara.

The left foot shows a partial synostosis of the metatarsal and cuboid bones and the same condition as of the fourth metatarsal as the right.

MICROSCOPIC HISTOLOGY.—Decalcification in trichloracetic acid, stain hæmatoxylin, eosin and Van Gieson. The topographic relations of the various formations of the

ossifying process to the epidermis are illustrated in Fig. 9, and present the extreme distance of the bone formation from the epidermis in any of my sections. To save space and expense, this relation is shown only where essential in the subsequent pictures.

Embedded in the distended and torn meshes of the corium are variously sized nests filled with red blood-corpuscles. Throughout these hemorrhagic masses a few more or less slender tendrils of the torn connecting tissues are remaining. The smaller of these masses show no appreciable changes discernible in the surrounding tissue. In the largest one sees greater tissue destruction within the hemorrhagic deposit and a slightly denser (pressure) approximation of a few connective-tissue fibres along the border encircling the hemorrhage.

There are few connective-tissue fibres within the blood extravasation, they are not so close together, the interstices between them larger, and around the margin of the hemorrhagic depots a circlet of the distended connective-tissue fibres. These fibres are the first indication of the local boundaries in the later ossification.

In these and in many of the later-stage sections a zone of badly stained or totally unstained tissue is observed surrounding the forming ossifications. I claim these to be areas of more or less impaired tissue nutrition caused from pressure of the initial hemorrhage.

In more advanced fields with large hemorrhagic central areas one observes a marked hyperplasia of the immediately surrounding connective tissue. There is a distinct oval shape of the border, forming a growing ring of densely crowded fibres and issuing a communicating network of branch lines throughout the hemorrhagic masses, dividing them into alveolar-shaped compartments. Sometimes these branches do not form and we encounter then in the later stages fully enclosed ossified rings enclosing homogeneous hemorrhagic centers. The progress of osteogenesis is from within outward, the region adjoining the erythrocytes representing the youngest stage. The erythrocytes apparently change into or stimulate the growth of fibroblasts. These cells are forming bone (layers) by apposition, the youngest toward the centre, the older outer ones gradually changing into osseous structure.

In a still more advanced stage, at a period preceding and during the beginning and progression of calcification, the outer encircling ring loses gradually its fibrous structure and becomes a lamellated homogeneous structure, with regular bone corpuscles and canals, or reaches this final osseous stage through an intermediate change from the fibrous to the cartilaginous tissue, in many instances during this metamorphosis the osseous border encloses firmly, and in many parts meets, without any intervening tissue or interior branches, the imprisoned mass of red blood-corpuscles.

This direct chain of transformation, however, is not the only one. In some parts we meet a single row of osteoblasts located nearly exclusively along the inner borders of the bone ring or situated in shallow lacunæ and contributing their share to the bone-formation in the outer ring and along the inner branches. They are very large mononuclear cells, the origin of which remained uncertain, perhaps conjoined transformed fibroblasts, as their gradual structural changes can apparently be followed in successive stages. The cartilaginous route finds its representatives at other places where a condensed connective-tissue mass around a primary hemorrhage changes, with the aid of the enclosed extravasation, from its fibrous condition to a homogeneous undifferentiated hyaline basal structure, with large cartilage cells of principally monocellular type imbedded therein. There the younger inner portion of the ring still shows cartilaginous tissue, while at the same location the adjoining outer part is changing into osseous structure.

This ossifying ring has in many instances an inside lining of single or multiple rows of fibroblasts. These fibroblasts crowd together and lose their identity in joining to build up the homogeneous mass. One sees in places an apparently gradual change of erythrocytes into fibroblasts, the precursors of the bone-tissue, small,

spindle-shaped nucleated cells, the youngest not larger than the red blood-corpuscles. They are lying at the inside of the ring between it and the central mass of unchanged erythrocytes, some, as possible witnesses of their new osteogenetic functions, still recognizable near the growing outer ring of bone tissue. At places there are finger- or fungus-shaped protuberances forming various sized and shaped projections from the outer ring into the hemorrhagic mass. At its inner irregular, ragged edge, blood-corpuscles and fibroblasts are fairly mixed in their genetic endeavor to serve as the building material for the new bone. Osteoblasts also take their part in the neoformation, but, as mentioned before, are nearly exclusively distributed at the inner side of the surrounding bone ring.

Other fibroblasts by crowding closely together form the ground substance in places within the central hemorrhagic mass for the new-bone islets and branches. Some of these are still interspersed with cartilage-like cells, which later, during the process of calcification, give way to the bone corpuscles and their ramifications.

New islets appear in many spots of the central mass. They grow and became confluent, and an intertwining network of new bone structure is formed extending from the inner side of the surrounding ring throughout the inner space. The fibroblasts leading the way for all this new structure extend cobweb-like bands developing into progressive stages of ossifying tissue, new-formed capillaries fill every interstice, and, as a resultant from united efforts, they gradually present the picture of bone and bone-marrow. In another stage the calcified connective-tissue ring around the blood extravasation has consolidated from the inside of the ring outward; the cells have lost their individuality; have formed one solid striated mass with the interspersed bone-cells; calcification and ossification have changed the fibroblasts into an elliptic more or less closed ring of bone tissue surrounding the remaining erythrocytes, which show various stages of apparent transformation. They are more or less closely connected with the inner surface of the bony ring, separated in some places from it by a very slender single layer of spindle-shaped cells; in others, touching it directly.

In these latter places the bone is still in the making, and so closely are the erythrocytes interwoven with the youngest spindle-shaped fibroblasts entering into the new bone-formation, that, contrary to all genetic doctrines, one could imagine the direct transformation of blood-cells into these bone-forming cells, which, in changing from the round- to the spindle-shaped form, become nucleated, growing gradually and less separable form in intermediate stages the ground substance, and, with the appearance of the bone corpuscles, the connecting link between the blood-cells and the finished bone tissue. At other places the above-described cartilaginous route is seen where the inside younger parts of the ring show cartilage tissue, while the adjoining outer ones have already changed to bone substance.

The fate of the central blood extravasation is a varied one. Simultaneously with the apparent participation of the blood-cells in the construction of the bone ring there is an energetic growth of interlacing branches extending from the inner side of the ring, forming a network of slender bands throughout the enclosed space. These branches are at first composed of one layer of spindle-shaped fibroblasts, suggesting their origin from transformed erythrocytes, and inclose in the meshes of the net a proportionate quantity of blood-cells from the original extravasate. Layer after layer is added to the first thickness of the branches, the structure of which with increasing volume, like the outer ring, becomes denser and undergoes the change into bone, forming a progressively continuous network of its own, which still encloses the remnants of the more or less changed erythrocytes. In other places of the sections, occasionally near initial hemorrhages (Fig. 40), some minute specimens of bone are seen, which are solid throughout and the ossification of which has apparently proceeded in adding fibro-osteogenetic layers, filling in the space within the outer border line with a finally solid tissue, thus eliminating the trabecular crosswork, but showing even at these stages in some places the enclosed remnants of the pioneer blood-extravasate, fibroblasts, or less changed erythrocytes.

This apposition from within, however, is not the only means of osseous growth. In a few places there also is a small aggregation of fibroblasts at the outside of the ossifying ring adding to its growth, but no osteoblasts appeared anywhere on the outside, showing that their location seems strictly limited to the inner aggregation of erythrocytes. In some sections a hypertrophic growth of outer connective tissue, participating in the ossifying process, may be seen to enter through a gap in the branches of the partly open osseous ring and closing it first with a connective-tissue bridge, which later ossifies, having previously sent a continuation of the ingrowing tissue from the gap toward the inner side of the ring. In others there is a predominant erythrocyte participation in the bone-formation of the ring.

We may even in some isolated instances get pictures that suggest a prevailing bone-formation from the outer side of the ring, the connective tissue changing directly to bone-tissue by calcification and development of bone-corpuscles and canals.

In these cases the inner erythrocytes play a minor part in the final ossification; the major part is taken by the connective tissue direct.

The structural changes of the hemorrhagic nucleus do not always keep pace with the development of the bony ring in all parts of an ossified nodule nor in every specimen of a series, and allowances have to be made for more or less of the erythrocyte-filling dropping out during the course of preparation, especially from paraffin sections. Thus we see pictures where a completely unbroken osseous wall closely surrounds a structurally, nearly unchanged, inner hemorrhagic mass. In others a more active participation of the connective-tissue growth in osteogenetic activity and a rather regressive metamorphosis of the enclosed hemorrhagic centre part.

In all these various formations the primary incentive is the hemorrhage with a consecutive connective-tissue growth, and whether the final result is a spongeoid or a solid bone, the route to it directly from the erythrocytes into ossifying fibroblasts or from those via fibrous connective tissue, hyaline or fibrous cartilage, with or without the help of osteoblasts, we have always to look back to the first hemorrhage as the starting point.

Analytical Study of Reported Cases

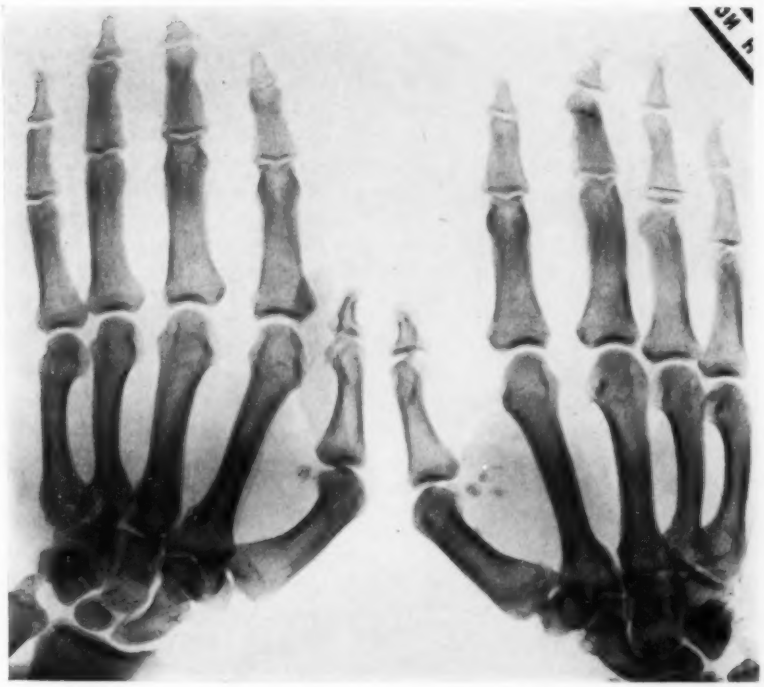
Before we proceed to analyze these 120 cases, we must explain that five of the number (four male and one female) were only entered in the list on account of their having figured in all the former compilations, and might be classified as "overlooked" by later investigators. They are the cases of E. Schwarz (No. 35), Kronecker (No. 36), E. Lexer (No. 50), Eichhorst (No. 53), and Krause and Trappe (No. 86). Neither of these is myositis ossificans progressiva, and they must be deducted from the total combined number (120-5-115). Four of these are males, one a female, so we have among 115 true cases 68 males, 42 females and 5 where the sex has not been recorded—a somewhat different proportion from former statistics. (Rolleston, 5 to 1; Nissim and Weill, 38 to 12.)

Their ages at the time when the first symptoms were observed were in the period before Muenchmeyer's classification (1868):

Males—0-5, 2; 5-10, 0; 10-15, 3; 17, 1; 18, 2; 21, 1. Females—8, 2; 5, 1; not recorded, 8. Not myos. prog., 5.

After Muenchmeyer's: Males—0-2, 19; 2-5, 23; 5-10, 7; 13, 2; 14, 1; 18, 1; 19, 1; ?, 1. Females—0-2, 10; 2-5, 17; 5-10, 10; 12, 1; 20, 1. Sex and history unknown, 1.

FIG. 1.—*a*, area of ossification in abdominal wall; *b*, central hernia.



R. FIG. 2. L.

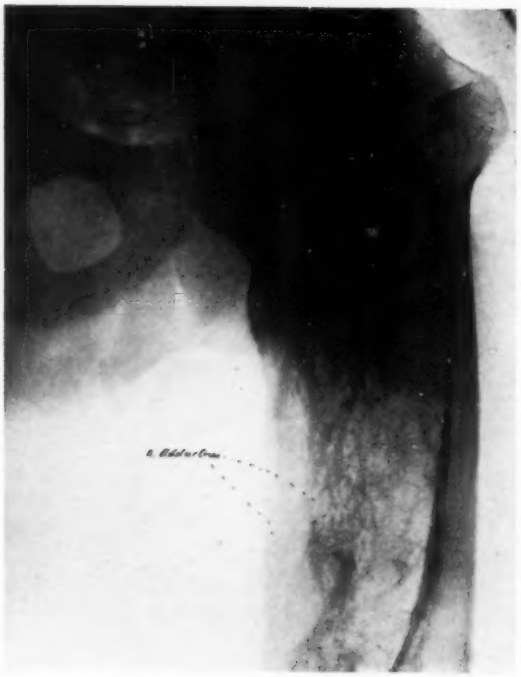


FIG. 3.

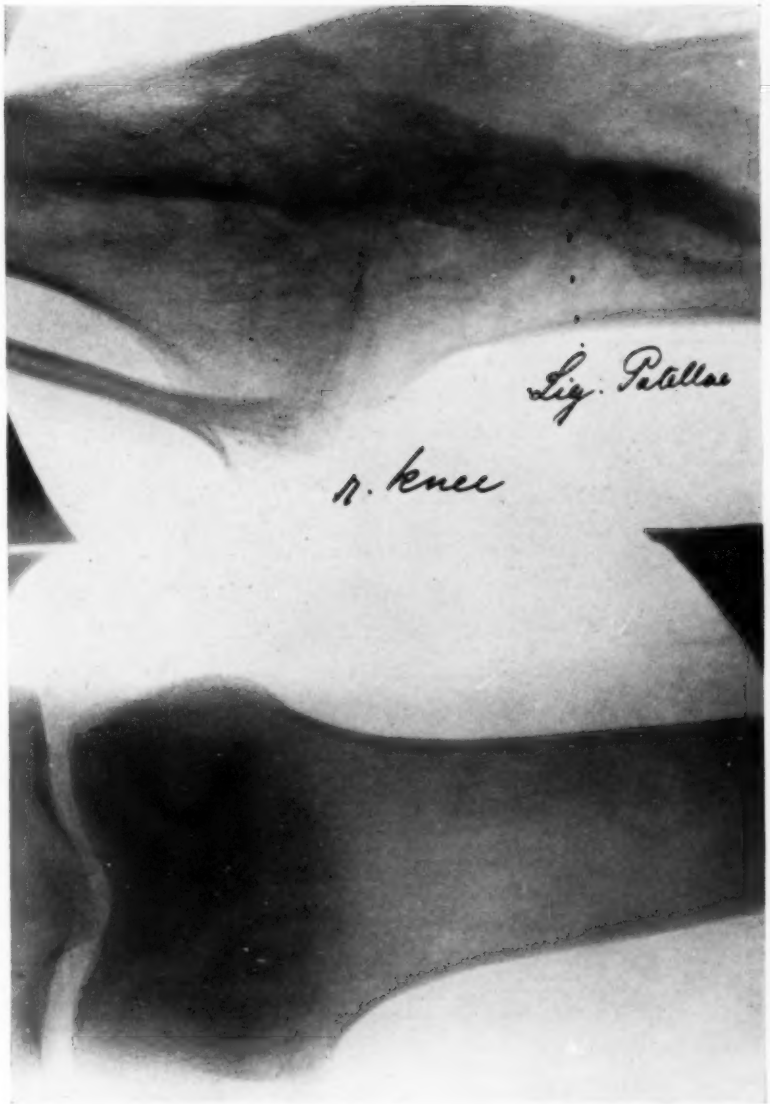


FIG. 4.

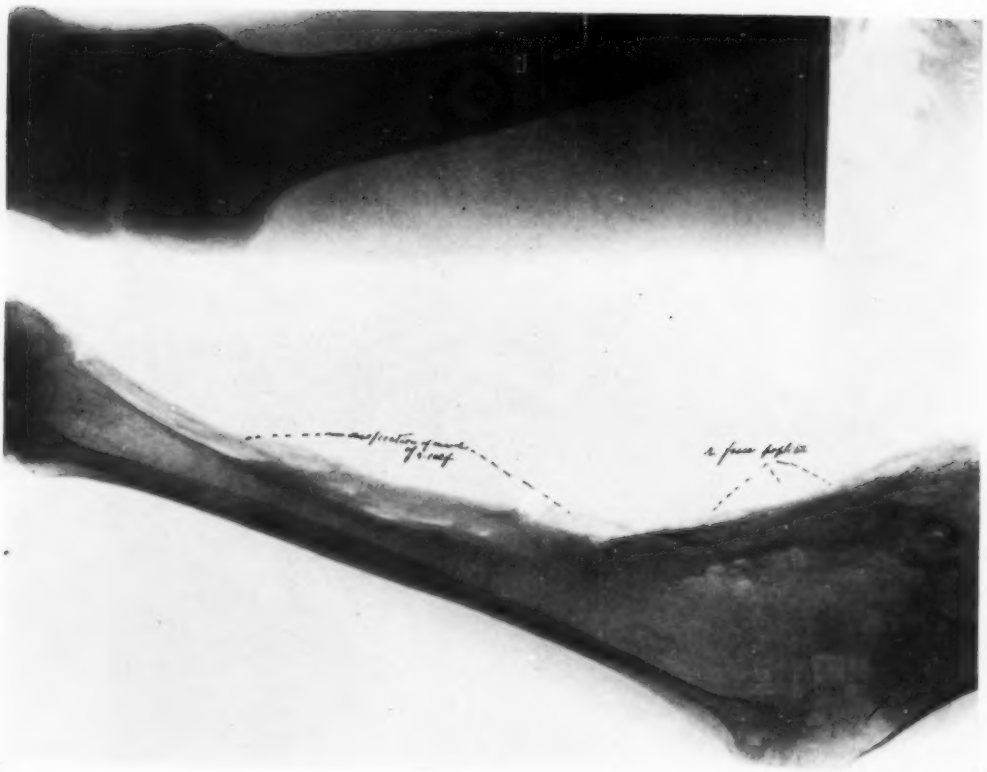


FIG. 5.

FIG. 6.

FIG. 7.

FIG. 8.

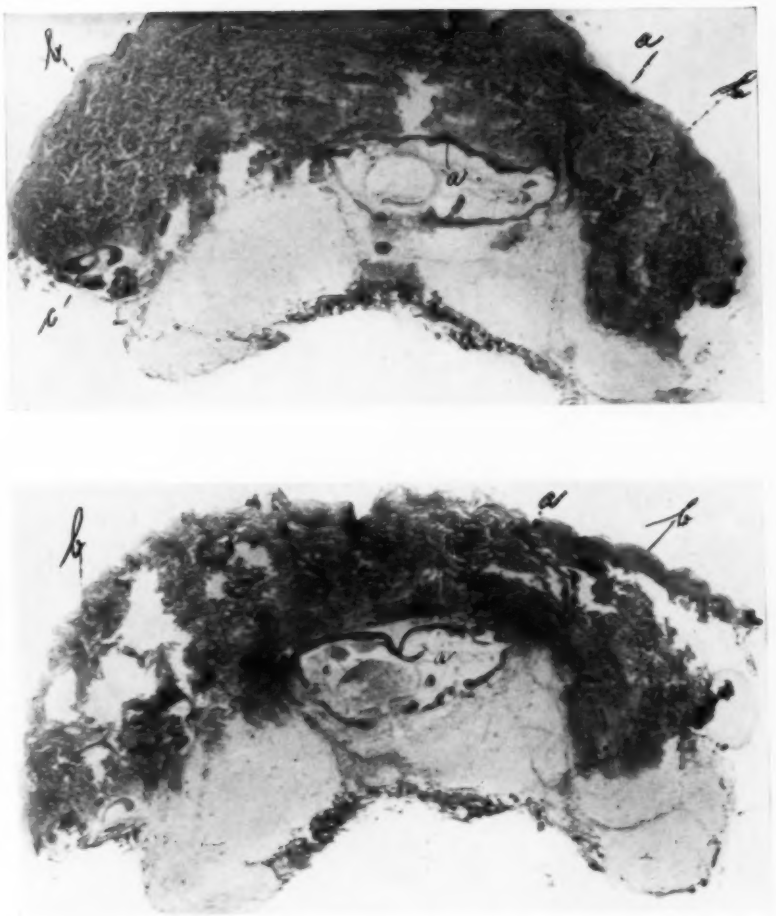
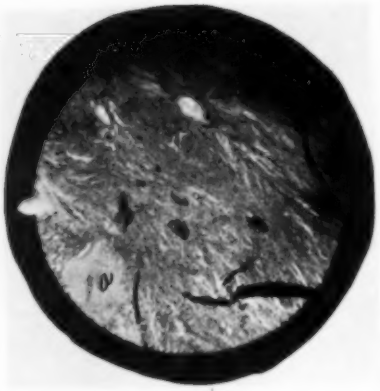
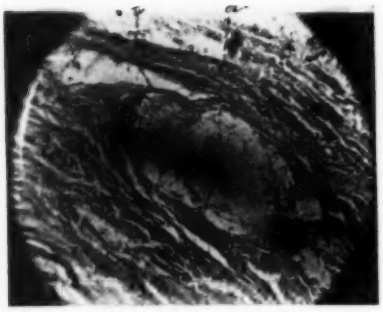


FIG. 9.—Topographic view of skin sections with bone ring. *a*, bone ring; *b*, epidermis.



A

B



C

FIG. 10.—The initial hemorrhage. *A*, low power (oc. II, obj. 16). *a*, hemorrhage (earliest stage); *b*, invasion of surrounding area. *B*, low power (oc. II, obj. a *). *a*, hemorrhage; *b*, epidermis. *C*, higher power (oc. II, obj. 4 ap). *a*, hemorrhage; *b*, connective tissue.

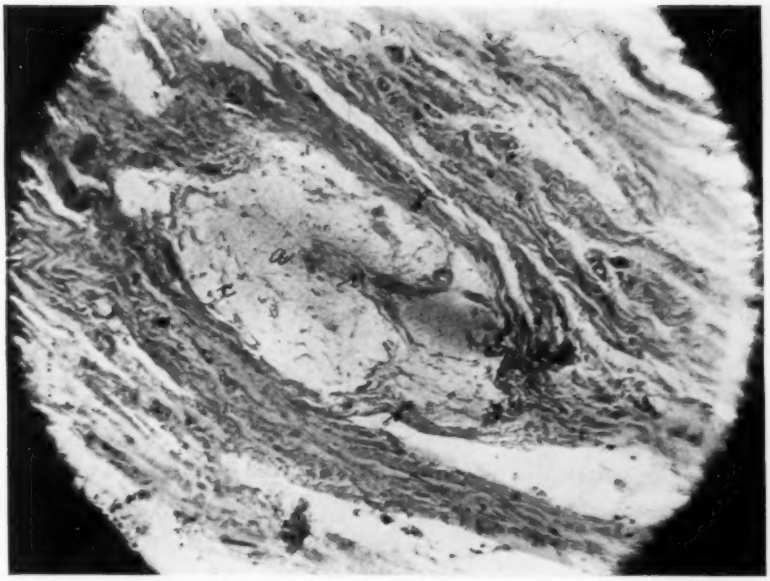


FIG. 11.—*a*, primary hemorrhage; *b*, beginning of boundary formation; *c*, displaced and torn shreds of connective tissue strands. Enlargement of Fig. 10*C*.

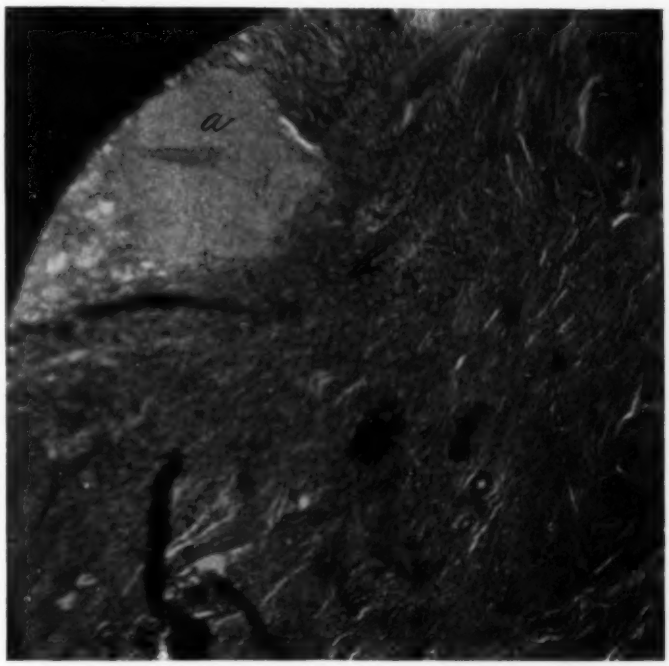
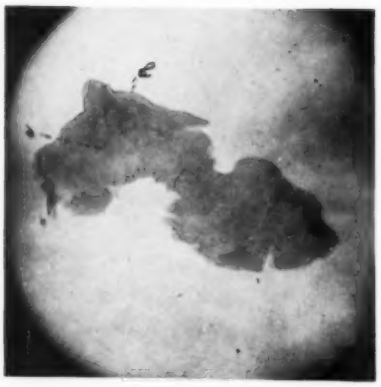


FIG. 12.—*a*, primary hemorrhage; *b*, invasion into surrounding normal tissue. Enlargement of Fig. 10*A*.

A



B

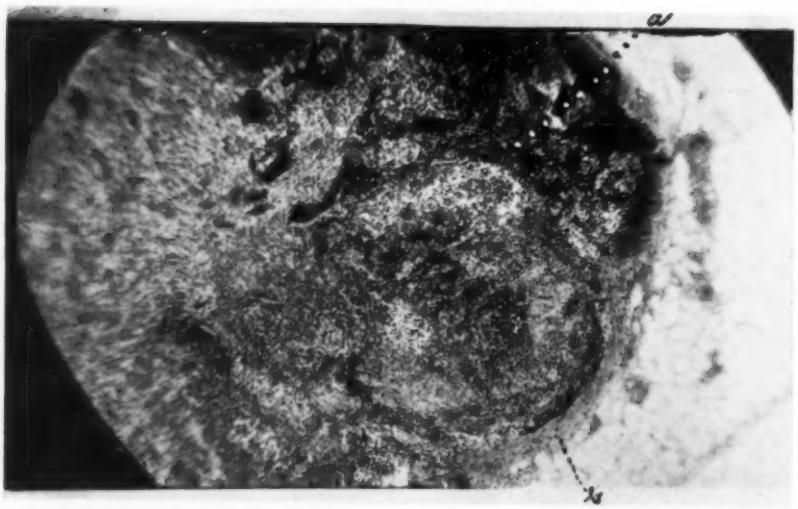
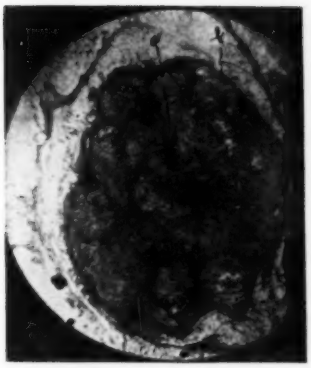


FIG. 13.—Initial hemorrhage with beginning encapsulation. *A*, low power (oc. II, obj. a *). *a*, hemorrhage; *b*, epidermis. *B*, higher power (oc. II, obj. 8). *a*, hemorrhage; *b*, beginning connective-tissue circlet.

A



B

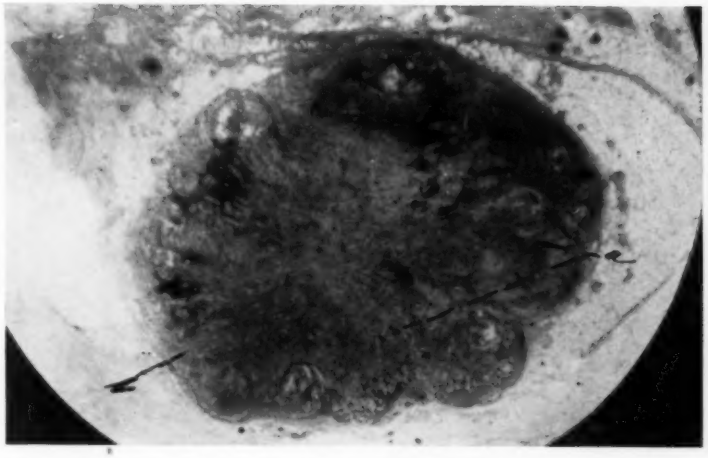
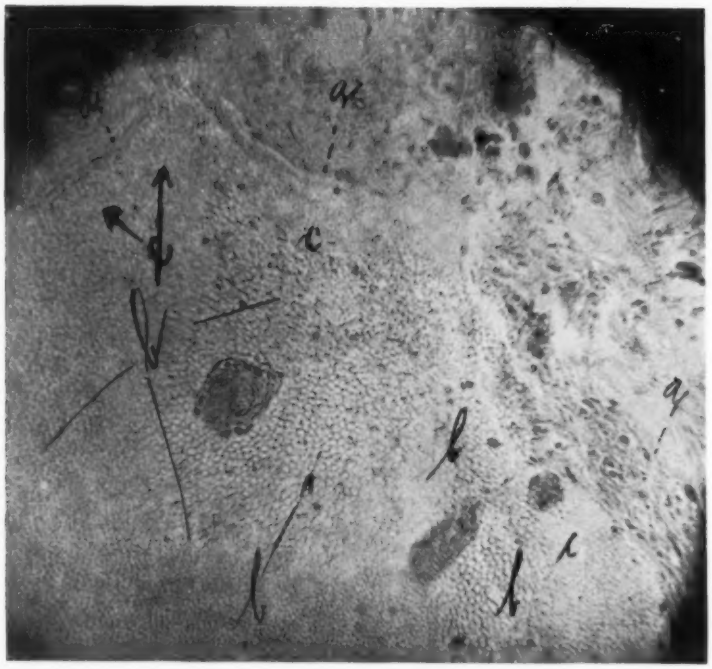


FIG. 14.—Initial hemorrhage with advanced connective-tissue encapsulation. *A* (oc. II, obj. 6). *a*, hemorrhage; *b*, connective-tissue circlet. *B* (oc. 5, obj. 8). *a*, hemorrhage; *b*, connective-tissue circlet.

A



B

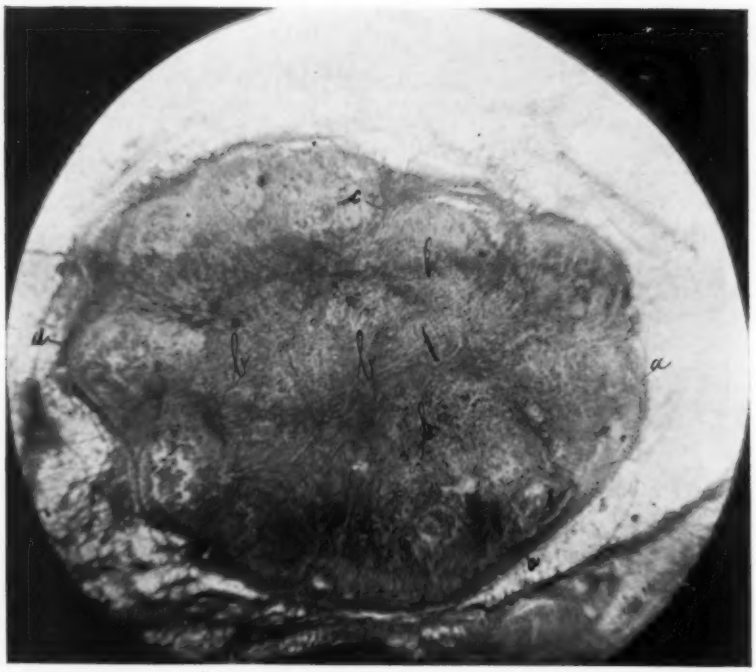
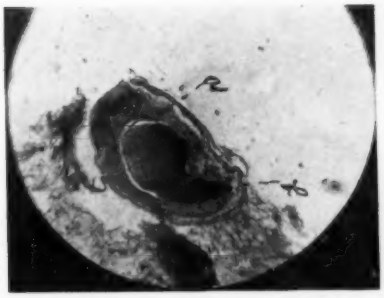


FIG. 15—*A*. *a*, beginning of ring formation; *b*, erythrocytes; *c*, change of form of erythrocytes to spindle shape. *B*. Ring formation nearly completed. *a*, newly formed ring; *b*, erythrocytes; *c*, slight projections from ring into erythrocyte mass.

A



B

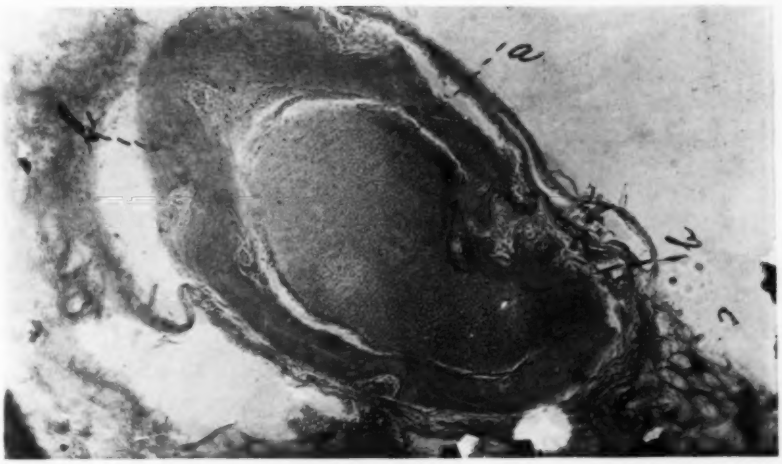


FIG. 16.—Solidification of outer connective-tissue ring, hemorrhagic centre. *A*, *a*, hemorrhage; *b*, solidified osseous ring (oc. II, obj. 16). *B*, *a*, hemorrhage; *b*, solidified osseous ring (oc. II, obj. 8).

A

B

FIG. 17.—*A*. Solid hemorrhagic mass in close apposition to ring in advanced stage of ossification (enlarged part of Fig. 17). *a*, bone ring; *b*, mass of erythrocytes; *c*, change of form of erythrocytes to spindle shape. Enlargement from part of Fig. 16. *B*. Solid hemorrhagic mass in close apposition to ring in advanced stage of ossification (enlarged part of similar section as Fig. 16). *a*, bone ring; *b*, mass of erythrocytes; *c*, change of form of erythrocytes to spindle shape.

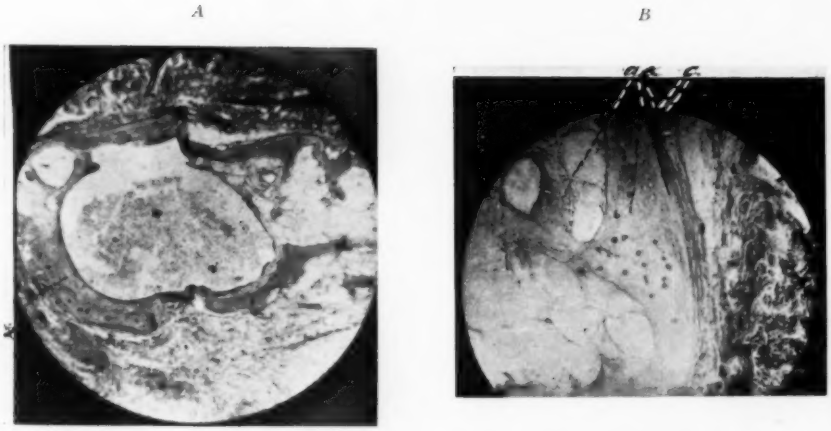


FIG. 18.—Cartilaginous tissue bordered on inside of ring by fibrous on outside by osseous tissue. *A*, *a*, place where magnified picture was taken (oc. 2, obj. 16). *B*, *a*, fibrous part; *b*, cartilaginous part; *b'*, fibroblasts changing into cartilage cells; *c*, osseous part (oc. 6, obj. 8). Enlargement of *A*, (*a*).

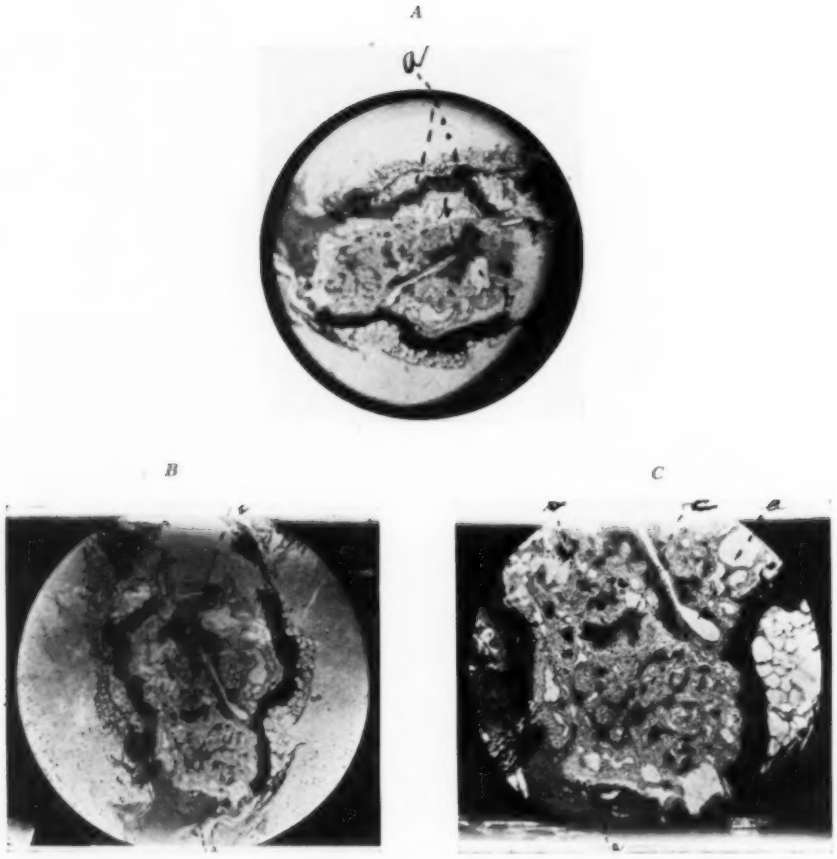


FIG. 19.—*A*. Osteoblasts on both sides of ossifying branches. *a*, outer ring; *b*, osteoblasts (oc. —,obj.—.). Osteoblasts on inside of ossifying ring and erythrocytes participation. *B*. *a*, osteoblasts inside ring; *b*, erythrocytes participation and apparent change (oc. II, obj. 8). *C*. *a*, osteoblasts inside ring; *b*, erythrocyte participation and apparent change; *c*, osteoblasts at branches.

A glance at these two groups shows the first observations of the initial symptoms at a remarkably earlier average in the second group than in the first. Although the number of the first is too small for a valid comparison, still the influence of Muenchmeyer's publication in drawing the attention of the profession to this mysterious disease cannot be ignored.

Since Helferich's enlistment of the congenital digital deformities among the symptoms of this affection their registration has not been followed by all authors. We believe that they are present in practically all genuine cases, and consequently of the ninety-three cases of this series the thirty-one where they have not been registered demand an explanation. In the first place, we have to deduct the five spurious cases already mentioned, and one of the other remaining (Bernacchi, No. 42) shows plainly on the accompanying photograph microdactylia of the thumbs. All of the other twenty-five who answered my inquiries (six) as to the presence or absence of these congenital deformities in their cases admitted that they had failed to look for them. In fact, all the thirty-one histories, with one exception, contain no reference to this symptom, and in my abstract of their cases I always inserted under the heading of congenital deformities, "not mentioned." There is only one author (Salvetti, No. 79) who specially dwelt *on the absence* of microdactylia of either fingers or toes in the description of his case, but his subject was very young (four years) at the time of observation, when slight differences in the size of fingers or toes may easily escape notice, and a decisive X-ray showing the condition of the epiphyses was not taken. Several of the remaining cases have as their only records brief demonstrations before medical societies. My conviction, therefore, that every genuine case of myositis ossificans progressiva shows mycrodactylia of either fingers or toes, or both, holds good in spite of the nineteen questionable cases. Speaking loudly in my belief in the failure of noticing this symptom stands the example of the great master of close observation, Rudolf Virchow, who failed to mention it in the history and description of his celebrated case (No. 46), while attention to the presence of microdactylia was called later by Gerhard in the discussion following the demonstration in the Berlin Medical Society. The malformations present in the cases were distributed as follows:

Cases after Helferich (1879): Thumbs and big toes and little fingers, 6; all fingers and big toes, 2; thumbs and fourth fingers, 1; thumbs and big toes, 24; big toes, 29; fourth and fifth toes, 1.

The very marked preponderance in this compilation of the big toes, they being alone or in combination with other terminal parts of the extremities the seat of the brachydactylic deformity, is shown by their being represented sixty-one times in the sixty-three available cases.

One does not wonder that a disease, so baffling in its course, from the first causes to its ultimate state, should invite the speculative as well as the patiently investigating observer to lift the obscuring veil and solve this embarrassing puzzle.

One of the first attempts to explain the mysterious process was the

theory of a retention of calcium in the organism due to a faulty metabolism which failed to excrete its quota of lime. This excess of lime in the fluids and the tissues was presumed, by its mechanical or chemical action, to exert an irritating influence and cause ossification.

H. Duvy, in Abernethy's case, was the first to examine the urine of these patients for phosphate of lime and found the quantity lower than the average. This was confirmed by Dittmeyer[1], Helferich[2], Pinter[3] and Partsch[4]. Duvy, Partsch and Pinter found in addition the excretion of urea normal; of uric acid, kreatinine and earth phosphates, diminished.

As all these investigations were confined to the urine alone, without regard to fæces or to exact account of the intake, they are valueless and must be rejected.

Very exact and complete analyses, however, have been made by Burgerhout[5] and Austin[6], both giving absolutely negative results. As Burgerhout's excellent work, besides being buried in a dissertation, is written in Hollandish, and Austin's has been published separately from Painter and Clarke's case, which served for the investigation, I shall give both their tables here in full. They are self-explanatory and Burgerhout's deductions are conclusive.

Burgerhout's comments on these tables are as follows: "According to the results of the examinations of our patients, no difference in the exchange of matter seems to be observable. The metabolism according to the figures certainly does not deviate from that of normal persons. Under ordinary circumstances the resorption of the necessary amounts of nourishment leaves nothing to desire. The greater increase in this patient's bone-tissue produces no typical alteration in his metabolism through the composition of food which at all times exercises a governing influence upon the phosphates

The daily diet during A and C was: 1.5 l. milk; 250 gr. bread; 100 gr. rice; 75 gr. butter; 50 gr. sugar, and one cup of Liebig's bouillon, calculated as follows:

	Albumen	Fat	Carb. Hydrates
1.5 l. milk	46.875 gr.	45 gr.	67.5 gr.
		2.5 gr.	150. gr.
250 gr. bread	21.875 gr.		
100 gr. rice	6.875 gr.	0.9 gr.	77.5 gr.
75 gr. butter	0.468 gr.	65.25 gr.	.....
50 gr. sugar	.....	.....	50 gr.
	76.2 gr.	113.5 gr.	345 gr.

Equal to 2782 calories, or about 32 calories per kgr. bodyweight. (Patient lying nearly motionless in bed.)

[1] Op. citat.
[2] Op. citat.
[3] Op. citat.
[4] Op. citat.
[5] Op. citat.
[6] *Journal of Medical Research*, 1907, vol. xvi, p. 452.

TABLE A

FIRST PERIOD URINE ANALYSIS

Date	Quantity	Sep W	Cl	P_2O_5	N	CaO	MgO	Kr	Body Weight
June 29....	1280	1016	4.268	1.805	8.996	0.228	0.076	0.177	68.7 kgr.
June 30....	800	1032	4.557	2.292	12.611	0.197	0.074	0.532	
July 1.....	560	1029	3.971	1.053	6.372	0.207	0.057	0.181	
July 2.....	1430	1020	6.759	1.837	9.810	0.262	0.073	0.220	
July 3.....	1180	1023	5.792	2.136	9.416	0.244	0.085	0.120	
July 4.....	1020	1023	5.192	2.025	6.569	0.235	0.091	0.451	68.3 kgr.
Analysis of fæces during entire period.....			30.539	11.148	53.774	1.373	0.456		
			0.047	15.814	4.291	11.369	0.627		
Total urine and fæces.....			30.586	26.926	58.065	12.742	1.083		

TABLE B

Hunger period: nothing taken but water, of which 4.1 l. was consumed.

URINE ANALYSIS

Date	Quantity	Spec. W	Cl	P_2O_5	N	CaO	MgO	Kr	Body Weight
July 5.....	2100	1012	6.108	1.785	8.526	0.179	0.074	0.130	68.3 kgr.
July 6.....	1210	1016	1.219	1.954	7.996	0.196	0.102	0.416	
July 7.....	1730	1014	1.677	2.197	8.910	0.398	0.106	0.294	65 kgr.
Analysis of fæces during entire period.....			9.004	5.936	25.432	0.773	0.282		
			...	4.689	1.856	3.218	0.188		
Total, urine and fæces.....			9.004	10.625	27.288	3.991	0.470		

TABLE C

LAST PERIOD URINE ANALYSIS.

Date	Quantity	Spec. W	Cl	P_2O_5	N	CaO	MgO	Kr	Body Weight
July 8.....	710	1026	2.237	1.260	8.300	0.402	0.086	0.499	65 kgr.
July 9.....	500	1030	2.729	1.205	8.176	0.266	0.062	0.678	
July 10.....	530	1031	3.469	1.102	7.984	0.241	0.044	0.521	.....
July 11.....	710	1019	5.206	1.441	9.164	0.258	0.048	0.481	
July 12.....	1070	1018	5.058	1.198	6.861	0.189	0.039	0.284	
July 13.....	1320	1018	5.279	2.086	9.203	0.211	0.059	0.437	67.3 kgr.
Analysis of fæces during entire period.....			23.978	8.292	49.788	1.567	0.338		
			0.056	11.808	2.956	8.296	0.529		
Total, urine and fæces.....			24.034	20.100	52.744	9.863	0.837	.....	

TABLE D

	Cl	P_2O_5	N	CaO	MgO
Total hunger period.....	9.004	10.625	27.288	3.991	0.470.
Deduct 4.1 water l. consumed.....	0.500	....	....	0.463	0.023
Real loss during hunger period.....	8.504	10.625	27.288	3.528	0.447

TABLE E

	Cl	P_2O_5	N	CaO	MgO
Total first period	30.586	29.962	58.065	12.742	1.083
Total third period	24.034	20.100	52.744	9.863	0.873
Difference	6.552	6.862	5.321	2.879	0.246
1.3 l. more water consumed during 1st period than in 3rd	0.159	....	....	0.147	0.017
Real retention in 3rd period	6.393	6.862	5.321	2.732	0.229

AUSTIN'S TABLES[7]

	Daily CaO in food	Daily CaO in fluid	Daily Total intake	Daily CaO in urine	Daily CaO in fæces	Daily Total output	Daily Balance
First period November 28. Average of two series of seven days each	.880 gr.	.366 gr.	1.246 gr.	.472 gr.	.787 gr.	1.257 gr.	.011 gr.
3rd series	.560 gr.	.012 gr.	.572 gr.	.425 gr.	.473 gr.	.899 gr.	.327 gr.

Substance taken poor in calcium: Roast beef, white bread, grapes, butter, oranges, chicken, spring water.

and alkalines whereupon it hinges in these cases. During the hunger-period, when this disturbing influence ceases, we see distinctly that this bone-tissue, by its increased quantity, comes more into the foreground, and how our patient, in this way living at the expense of his body-tissues, also uses more of his increased amount of bone-tissue than normal persons." Austin's exact tables show a similar result.

These tables, representing a complete analysis of the entire intake and the total output through urine and fæces during three series of seven days each, show absolutely normal conditions and no retention of lime.

With the multiplicity of cases and with modern methods the pathological anatomy of progressive ossifying myositis has been fairly well cleared. No longer prevails the old definition of Virchow[8], who classified the pathological products as belonging under the heading of multiple osteomata and that the swellings representing such distinctive tumors always originate from the periosteum.

This view had also been expressed by Eberth[9] subsequent to his findings at an autopsy of a case of myositis ossificans progressiva which showed the ends of the muscles and their tendinous insertions ossified, the bodies of the muscles though relatively intact. This opinion was, moreover, shared at a much later date by Pincus,[10] who denied to our malady the distinction of being a type *sui generis*, adding that it belonged by all means to the class of osteomata and exostoses and possessed the indubitable characteristics of a tumor-disease, its original starting-point being

[7] A. E. Austin; Calcium Metabolism in a case of Myositis Ossificans; *Journal of Medical Research*, vol. xvi, March to July, 1907, p. 451.

[8] Die krankhaften Geschwuelste and op. *citat.*

[9] Ueber multiple Knochengeschwuelste, Deutsche Klinik, 1862, No. 9, p. 91.

[10] Op. citat., 1897.

the periosteum, and its casual element the hemorrhage from traumatism during birth.

Mays[11] was inclined to compromise. He asserts the pathological process to be in the osseous system along the course of tendons and fasciæ and in the intramuscular connective tissues as well as in the loose connective tissue between the muscles; but he affirms at the same time that the entire tissue complex is not subjected to this change, this anomaly being nowhere encountered in the subcutaneous tissue, affecting only the connective tissue of that apparatus which Virchow has singled out as a seat of the multiple osteomata, "the apparatus of motion."

He was disposed to range this disease with the exostoses, although his microscopical examinations point toward an adoption of the theory of a congenital predisposition to the ossification of the intramuscular connective tissue, the fasciæ and aponeuroses.

Muenchmeyer[12] was the first to pronounce this view, and in separating it from all cases of multiple osteomata he drew a concise picture of the malady and characterized it as a peculiar and distinctive constitutional anomaly.

Cahen[13] pronounced, as the first, an absolute identity in the microscopical pictures of traumatic and progressive ossifying myositis and a direct causal connection between the hypertrophy or the intermuscular connective tissue and ossification. Other reliable microscopical observers confirmed, in more or less complete form, these findings.

Lexer[14] asserts that the excessive increase of the intramuscular and connective tissue-cells and the formation of an indifferent tissue, rich in cells and profusely vascularized appearing with a simultaneous atrophy of the muscular tissue, is the forerunner of ossification. These new connective-tissue cells gradually assume large round forms and then change into cartilage cells.

Kraske[15] believes that those osseous neoformations, not emanating directly from the bones, are ossifications extending from the insertion of the muscles into the tendon-fibres and fasciæ and the connective tissue of the muscles. The muscles do not participat in this change, and waste away through pressure-atrophy and fatty degeneration.

In Goto's[16] very extensive microscopical studies of his case the author arrives at the conclusion that the primary process is the hyperplasia in the fascia or aponeurosis, tendon or periosteum, also, under certain circumstances in the ligamentary apparatus.

The muscular interstitial tissue, which most authors describe as the primary seat of the affection, is, according to his investigations, only secondarily invaded from the neighborhood. He would call the disease logically hyperplasia fascialis ossificans progressiva. He divides the course of the disease into three stages:

1. The stage of connective-tissue hypertrophy without inflammation.
2. The stage of fibrous induration.
3. The stage of ossification.

The new-formed bone always shows a normal bone structure.

In this paper Goto attaches no importance to the hemorrhagic extravasations found in all of his specimens excised at an early stage of the local affection. Pieces excised later still showed traces of an earlier hemorrhage, but mostly absorbed. He expresses the belief that the hypertrophy of the connective tissue takes place so rapidly and energetically that the muscular elements in spite of their elasticity have no time

[11] Op. citat.
[12] Op. citat.
[13] Op. citat.
[14] Op. citat.
[15] Op. citat.
[16] Op. citat.

to escape the process of distention. They, together with the thin-walled blood-vessels, are finally torn, and thus cause the secondary hemorrhagic foci. We will see later that his experimental work for traumatic ossifying myositis shows another aspect of the importance of blood and blood-vessels for the ossifying process.

The microscopical findings in my own case, while confirming that the primary tissue change takes place in the extra and intramuscular connective tissue, add to this a hitherto unknown feature of this disease, a bone-formation in the skin—a feature contrary to all previous experience. It involves principally the corium and therefore does not definitely trespass into the ectoderm; but its independent and isolated occurrence, in even the connective-tissue layer of the skin, has not been observed before. This find more than any other proves the primary tissue affected in this malady to be the connective tissue of the mesoderm, and the secondary and passive part, the muscular tissue. The significant presence of hemorrhagic extravasations as forerunners and foci of the new-formed cartilage and bone will be considered in connection with the question of the immediate cause for the bone-formation.

In considering the various causes theoretically or experimentally advanced for the explanation of bone-formation in this disease, I shall also include those for the *traumatic* myositis ossificans. While those based on the trauma itself can be easily excluded, we must recognize that the various stages in the microscopical pictures of the process of ossification are identical in both and may be profitably examined and reviewed in common.

The authors writing on this subject seem, with few exceptions, to be divided into two camps, one ascribing the stimulating cause to the presence of lime deposits, the other to blood extravasation into the tissues, due to the rupture of thin-walled new-formed blood-vessels. There is lacking in both explanations the ulterior reason or proof for the presence of lime or of the free blood, the theory of metabolic lime retention being untenable in the face of the exact analyses of Burgerhout and Austin, and except in the cases of traumatic ossification none either for the extravascular blood.

After a series of experiments and histological work, Berlies[17] arrives at the conclusion that the muscular osteoma (rider's bone) is not a tumor, but is formed in connection with the tearing off of a flap of periosteum. This is the basis of the new formation of bone, the detached flap surrounded by the muscular fibres acting as a bone-graft within the body of the muscles.

A development of bone tissue may also be gained by direct transformation of cartilaginous embryonic connective tissue, or of fibrous connective tissue.

Rathcke[18] communicates an interesting case bearing upon this latter phase of development. A sailor 28 years old was hurt by the fall of a sailing yard on his right thigh. A swelling resulted immediately afterward, which gradually hardened.

[17] Etude histologique et expérimentale des Ostéomes musculaires. Arch. de méd. expériment. Paris, 1894. Tome 6, p. 600.

[18] Ueber die Ursache d. gelegentl. Auftretens v. Knorpel bei d. Myosit. ossif. Arch. f. Entwicklgsmechanik d. Organismen. Bd. vii, 1898, p. 398.

Extirpation some time later of this hardened mass showed an intersection of the vastus internus, with bands of interstitial connective tissue growing harder the nearer it approached the bone. In the peripheral parts the growth was a cartilaginous tissue, but near the bone it was osseous and had to be separated with a chisel. Rathcke gives the explanation that where free motion exists, the tissue has to yield to changes of location and friction of the various tissue layers among each other and the cartilage adapts itself to these conditions, whilst the nearer the bone the traction and pulling is more and more prevented by its immovability, and only those cells survive which can offer the necessary resistance by their osseous transformation. This illustrates also the theory of Roux.[19]

Cornil and Condray[20] grafted entirely detached pieces of periosteum into the leg muscles of young dogs and produced thereby growth of osteomata in these muscles. The microscopical study of these and of extirpated human myosteomata convinced them that the bony tissue of these tumors grows at the expense of the connective tissue and not at that of the more or less transformed muscular tissue.

John Morley's[21] experiments on rabbits by scraping the periosteum and crushing the overlying muscle, produced bone-growth into the muscles. His specimen and X-ray pictures show this new growth to emanate exclusively from, and remain in direct continuation with, bone, forming an exostosis extending into the muscular tissue.

Another theory that some component of the joints, evidently the synovia, plays an important part in the ossification of myositis ossificans traumatica, was advanced by Ewald.[22] The lesion produces a tear in the capsule, some of the synovia escapes and then exercises the peculiar stimulus leading to ossification.

The experiments of Fabris[23] were based upon similar suppositions. His observation was that most cases of myositis ossificans traumatica occur in the neighborhood of articulations. He injected synovia into the muscles of dogs, but his results were absolutely negative; not the slightest trace of osseous tissue could be found in the injected area.

Among those who advocate that the presence of lime is the important cause of ossification, Cohn[24] believes that in a fair number of cases a chronic mechanical irritation by lime alone may lead to ossification (in blood-vessels).

Marchand[25] asserts that genuine bone may be produced by connective tissue everywhere where sufficient lime salts are available, *i.e.*, where calcification has preceded. Without the presence of lime no bone could be found, in spite of the most energetic hyperplasia of connective tissue.

Rohmer[26] believes that the first condition for the production of bone is the new formation of a highly vascularized connective tissue, and the second the presence of lime.

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[19] Gesammelte Abhdlgn. Ueber Entwickelgsmechanic, Bd. ii, No. XVIII. Leipzig, 1895.

[20] Sur le mode de dévelopement des ostéomes musculaires d'origine traumat. Bulletin de l'Académie de méd. Paris, 1907. Pp. 597–600.

[21] Traumatic intramuscular ossification. *British Medical Journal*, Dec. 5, 1913, p. 1475.

[22] Zur Aetioligie d. Myos. ossif. traumat. Zentralbl. f. Chir. 1910, No. 22, p. 771.

[23] Fabris: Sulla genesi della miosite ossificante. *Gazetta degli Ospedali e della Cliniche*, 1911, No. 42, p. 443.

[24] Cohn: Ueber Knochenbildg. in d. Arterien. Virch. Arch. Bd. 106, 1886.

[25] Marchand: Verhdlgn. d. deutsch. Pathol. Ges. Ber. ueb. d. 2 Tag. Aachen 17-20, September, 1900.

[26] Ueber Knochenbildgn in verkalkten endocarditischen und endarter. Heerden Virch. Arch., Bd. 166, p. 13.

A similar view as to lime has been entertained by Pollak[27], who observed cases of heterotopic bone-formation in the lungs. It always occurred around calcified necrotic parts. His case (No. 21) showed an additional ossification of the stylohyoid; case No. 24, exostoses on the lumbar vertebræ.

Roepke[28] joins in the conclusions that the presence of lime salts within a traumatically inflamed connective tissue furnishes the irritation and cause for bone-formation.

Georg B. V. Gruber[29] emphasizes again the absolute identity in the microscopical pictures of slides of traumatic myositis ossificans cicumscripta and myositis ossificans progressiva, and adds that wherever young and very vascular connective tissue meets with previous lime deposits an osseous formation is possible without any relation to periosteum. Such has been observed in blood-vessels, lymphatics, glands, brain, liver, kidneys, vas deferens, etc. He also mentions the possibility of secondarily encysted blood or lymph deposits being the source of lime for bone-formation. In a previous paper[30] the same author arrived, among others, at the following conclusions: "That the periosteal genesis in myositis ossificans traumatica is improbable and is absolutely excluded except in cases where the muscle-bone is in direct communication with the skeletal bone; and that the ossifications of the muscles are caused by the united action of young cellular connective tissue meeting the deposits of calcium, or of being stimulated to ossification by its presence in the tissues.

"In order to decide the problem of the genesis of muscular ossification or to solve that of heterotopic ossification in general, the questions regarding the calcium metabolism and the calcification of tissues must be answered. Until now the acceptance of a certain individual disposition to such ossification cannot be dispensed with entirely."

Dibbelt[31] drew attention to the difference in the forming of bone between carnivorous and herbivorous animals, dogs instancing the difficulty among the carnivorous, rabbits the case of the herbivorous. He attributed these diversities to the high calcium metabolism of the herbivores, and supported this theory by animal experimentation. He fed rabbits on food poor in calcium and caused the formation of fibrous tissue in place of bone, the addition of calcium to the food effecting a metaplastic change of tissue into bone.

That free blood acts as a factor in the bone-producing process has been very frequently and persistently claimed by many authors.

Cahen[32] claims this in his paper where he asserts as the first, that the microscopical picture of myositis ossificans traumatica is identical with that of progressive. In his case the hemorrhage caused by the trauma marked the place of origin of the later ossification.

E. Cordillat[33] reports the case of a young soldier, 21 years, entering a cavalry regiment, who, between November 24, 1894, and January 19, 1895, rode altogether about fifteen miles, and who presented himself on the latter date with an ecchymosis, the size of a dollar, on the left leg posteriorly, and a hard, bony mass at the inside of the femoral vessels occupying the entire middle adductor, a length of 17 cm., with

[27] Ueber Knochenbildgn. in der Lunge. Virch. Arch., Bd. 165, p. 129.

[28] Z. Kenntn. d. Myositis ossif. traumat. Arch f. klin. Chir., Bd. 82, p. 81.

[29] Weitere Beitraege z. pathol. Anat. d. umschrieb. Muskelverkng, nebst Bemerkgn. z. Myositis ossif. ueberhpt. Mitth. a. d. Grenzgb. d. Med. and Chir., 1914, Bd. 27, pp. 762-786.

[30] Ueber Histol. & Pathogen. d. circumscript. Muskelverkng. Jena, G. Fischer, 1913.

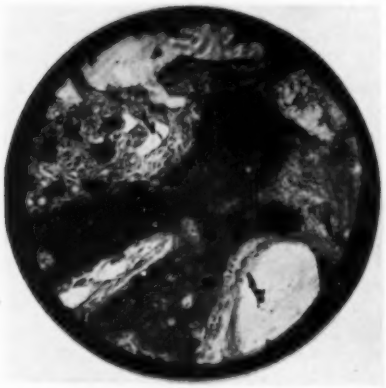
[31] Beitraege z. Histogenese d. Skelettgewebe & ihrer Stoerungen. Beitraege z. pathol. Anal. & z. allgem. Pathologie, 1910. Bd. 48, p. 147.

[32] Ueber Myos. ossif. Deutsche Zeitschr. f. Chir., Bd. 31, 1890, p. 372.

[33] Ostéome bilatéral du moyen adducteur. Arch. d. Méd. et de Pharmac. militaires, 1895. Tome, 26, p. 427.

FIG. 20.—Osteoblasts participating in bone formation on inner side of ring and along branches. *a*, bone ring; *b*, osteoblasts (enlargement of Figs. 19 and 30 *A*).

A



B

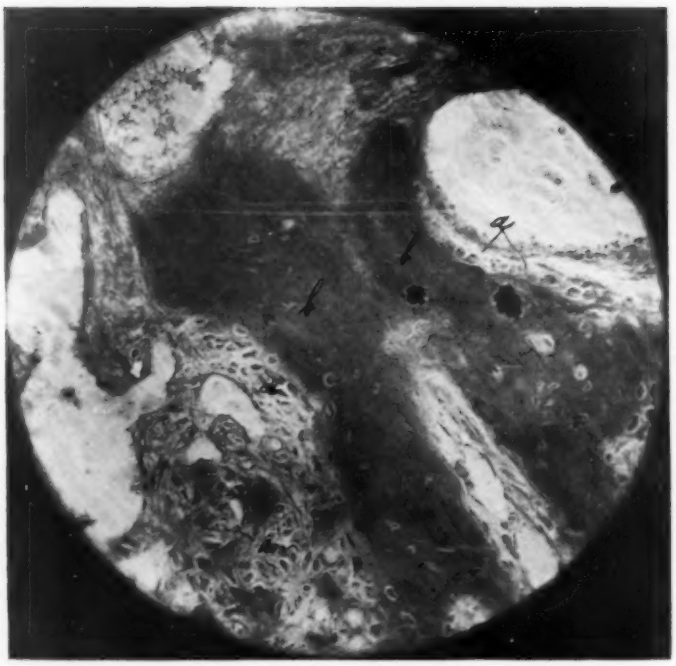


FIG. 21.—*A*. Osteoblasts on both sides of ossifying branches. *a*, branches (enlargement of Fig. 19); *b*, osteoblasts. *B*. Osteoblasts on both sides of ossifying branches (enlargement of Fig. 21*A*). *a*, osteoblasts; probable origin of osteoblasts from conjoined transformed fibroblasts. *b*, branches.

FIG. 22.—Inner part of ring cartilaginous outer osseous—Erythrocytes aiding bone formation. *a*, erythrocytes apparently entering bone formation; *a'*, erythrocytes in close opposition to youngest layer of ring changing shape to spindle form; *b'*, cartilaginous spot; *b*, bone ring.

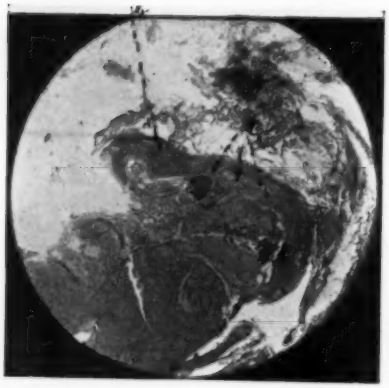


FIG. 23A

FIG. 23B

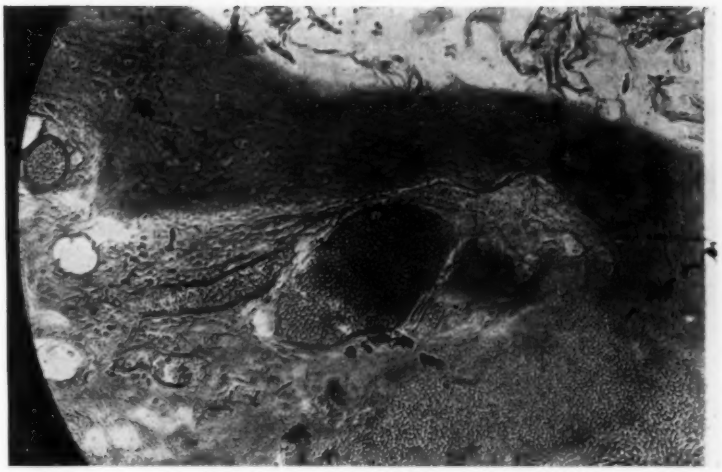


FIG. 23.—Erythrocytes apparently entering into bone formation and remainder of cartilaginous tissue next to osseous formation in ring. *A*, *a*, erythrocytes apparently aiding bone formation; *b*, cartilaginous spot (oc. II, obj. 16). *B*, *a*, erythrocytes apparently entering bone formation; *b*, cartilaginous spot (oc. VI, obj. 8). (Enlargement of *A*.) *C*, *a*, erythrocytes apparently changing into fibroblasts; *b*, cartilaginous spot (oc. VI, obj. 4).

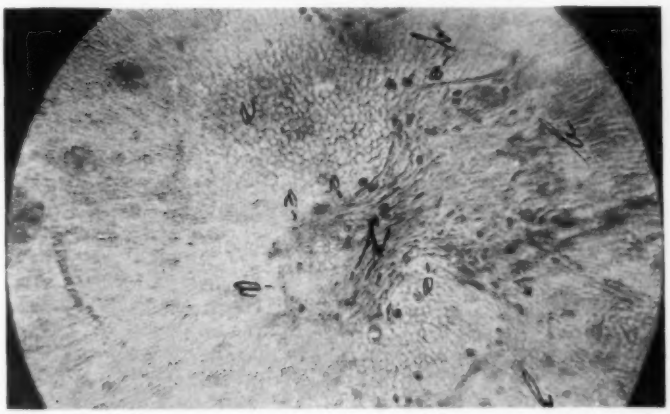
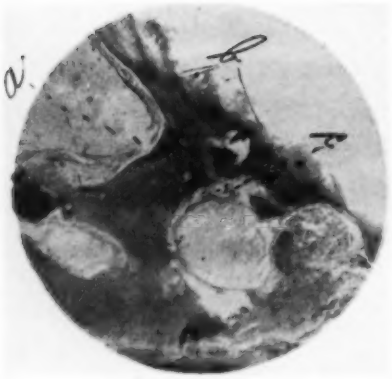


FIG. 24.—Apparent erythrocyte participation in bone formation of branch and ring. *a*, erythrocytes; *a'*, change of form of erythrocytes to spindle shape as they get into contact with the youngest osteogenetic layer of the ring and branches; *b*, bone ring; *c*, branch (very early stage).

A



B

FIG. 25.—Fungus and finger-shaped inner branches with osteoblasts (*a*) and apparent erythrocyte participation in forming branches and ring (*b* and *c*). *A*, *a*, branches; *b*, ring; *c*, osteophytes (oc. II, obj. 16). *B*, *a*, erythrocyte in branches; *b*, erythrocyte in ring; *c*, beginning formation of branch; *d*, areolar tissue (oc. II, obj. 4); *e*, as ostroblasts.

A

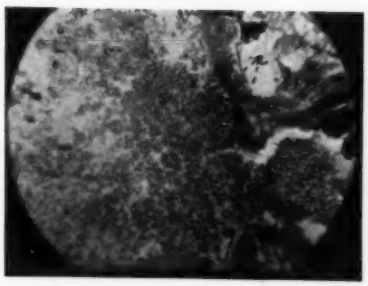
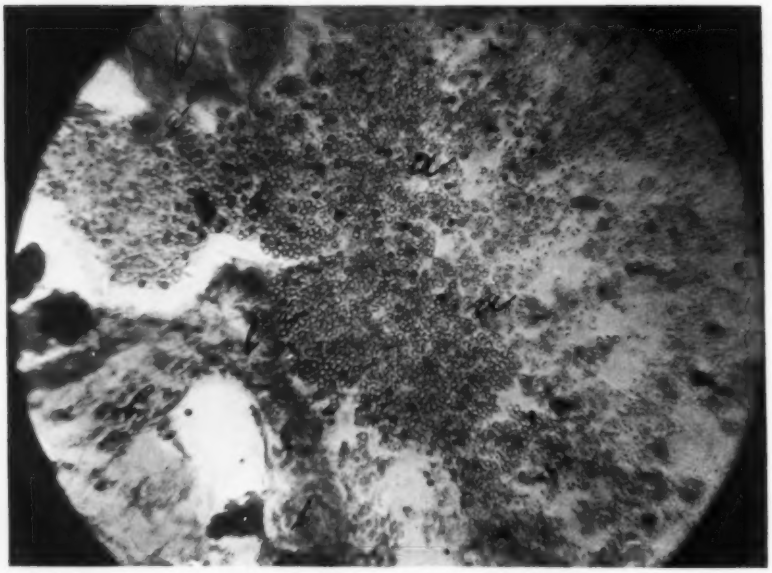


FIG. 26.—*A*, Apparent erythrocyte participation in building of branches in very early stage of formation.

B



C

FIG. 26.—*B*. Apparent erythrocyte participation in building of branches in very early stage of formation (enlargement of Fig 26 *C*). *a*, erythrocytes; *a'*, same entering into building of branches; *b*,'branches in very early stage of formation. *C*. Apparent erythrocyte participation in building of ring and branches in early stages of formation somewhat later than Fig. 26 *B*. *a*, erythrocytes; *a'*, erythrocytes in apparent participation of building of ring and branches in early stage of formation; *b*, ring.

a spur about 3 cm. long at its upper part. He was under daily observation until February 15, 1895, when the surgeons discovered an osteoma in the right adductor medius. It had formed unbeknown to the patient, occupying the same position as on the left and evidently from the same cause.

Duems[34] expresses his belief, founded on the observation of his cases, that the irritation of the connective tissue from the hemorrhagic infusion following the trauma is responsible for the ossification.

Bier[35] voices a similar opinion, accusing directly the hemorrhage of being the irritant for bone-formation.

Stempel[36] excised pieces of muscles from a case of myositis ossificans progressiva. His microscopical examination of the early stages always showed a hypertrophy of the fibrillary tissue separating the muscular fibres and an intramuscular hemorrhage, and although he never detected an increased cell infiltration or primary hypertrophy without hemorrhage, this author expresses the opinion that the excessive connective-tissue growth as the primary factor in the process causes the hemorrhage by pressure and later the ossification.

Steinert[37] observed ossification of muscles after hemorrhages into them, with patients whose limbs were paralyzed from cerebral apoplexy. In some of these cases the connection between hemorrhage and ensuing ossification is most striking, in a few others only very probable. In one of the cases a disposition to hemorrhages existed.

In their experimental work Sacerdotti and Frattin[38] found a nearly constant bone-production with bone marrow in kidneys of rabbits after ligation of renal blood-vessels, and consequent hemorrhagic necrosis and ossification throughout the organ. The necrotic tissue is riddled with blood-extravasations, and these give rise to ossification.

Berndt[39] found, in a free ossified muscular tumor extirpated twelve days after a trauma, a hemorrhagic extravasation entirely permeating the muscle and periosteum.

In his two excellent monographs on myositis ossificans progressiva[40] and myositis ossificans traumatica Goto[41] pleads for the recognition of an essential difference in the part taken by the hemorrhagic extravasation of changing into the ossifying tissue of the former disease from that in the latter. Although encountering in all of his early excisions of invaded tissues, either of the progressive or the traumatic, or of the experimentally produced forms of myositis ossificans, the characteristic hemorrhagic extravasation into the affected territory, he considers the hemorrhage of the progressive form a secondary feature induced by the hyperplasia of the connective tissue, which is supposed to finally tear the muscular fibres, together with the small, thin-walled blood-vessels, and thus cause a secondary hemorrhage, an explanation already advanced by Stempel.[42] In the traumatic form, however, Goto

[34] Deutsche militaeraerztl. Ztschr. 1887. No. 16, p. 325.

[35] Beobachtungen ueber Kochenregeneration. Arch. f. kl. Chirurgie. Bd. 100, p. 91.

[36] Op. citat.

[37] Ueber d. sogen. Myos. ossif. & verw. pathol. Verkngsproc. insbes. bei org. Nervkrkhtn. Mithlg. a. d. Grenzgeb. d. Med. & Chir. Bd. 21, 1910, p. 513.

[38] Sulla produzione eteroplasto dell osso Ricerche sperimentali. Giornale della Academ. di Med. di Toreno. Vol. vii, 1901; fasc. 12 & Virch. Arch. 1902, Bd. 168.

[39] Arch. f. kl. Chirurgie. Bd. 65.

[40] Patholog. & Anatom-klin. Studien ueb. d. sogen. Myositis ossif. progr. Arch. f. klin. Chir. Bd. C, p. 730.

[41] Die sogen. Myositis. ossif. circumscripta traumatica. Eine chirurg. experimentelle Studie Mitthlgn. aus. d. mediz. Fakultaet d. Kais. Univ. Kyushu, Fukuoka, Japan, Bd. 1, 1914.

[42] Op. citat.

recognizes the primary part of the hemorrhage, and the microscopical examination showing no difference between the two, he considers hemorrhages in the latter the primary cause and essential for bone-growth in consequence of the trauma; and although he constantly found their traces as well in fresh attacks of myositis ossificans progressiva, they are considered by him as a secondary trait only in this disease.

Goto employed for his experimental production of myositis ossificans indirect traumatism of the periosteum by hammer blows upon a portion of the femur, only thinly covered by muscle, and transplantation of periosteum into the muscular substance by either pedicled or free flaps, or of fine pieces chopped in physiological salt solution. They were all successful. He also tried hammer blows, and other traumatisms, with and without intramuscular injections of small quantities of blood, and finally the method of G. Koga[43]—repeated intramuscular injections of 1 per cent. solution of chlorate of lime, 3 cm. to the kgr. weight of rabbit. Of this last series of experiments, solely the ones with chlorate of lime injection showed ossification centres; the ones with the injections of blood alone showed calcification only.

The excisions and examination of these latter took place only thirteen days after the injections, whilst those of the chlorate of lime series were allowed to stay thirty and more days. The question if a longer interval between injection and excision of the blood injections would have given results of ossification must remain unanswered at the present time. The same experiments on guinea pigs showed early X-ray shadows.

Duems-Vulpius[44] approach this quest from an experimental as well as clinical standpoint. Their experiments and their casual observations of these ossifications always showed hemorrhagic extravasations as their precursors.

Among the latest publications on heteroplastic ossifications, two notable papers, Moshkowitz[45] "On the Relation of Angiogenesis to Ossification, etc.," and George W. Outerbridge's[46] "Non-teratomatous Bone-Formation in the Human Ovary," require special attention. Moshkowitz quotes the older Rabl-Schulte doctrine of the specificity of endothelium, which takes it from the series of mesenchymal derivatives and separates it absolutely from the blood should this prove to be of mesodermal origin. Consequently, thinkers who support this view have seen the importance of assigning an entodermal origin to both blood and endothelium. He then endorses the modern theory of angiogenesis (first proposed by Rueckert and Mollier) that blood-vessels form from clefts and spaces within the mesenchyme bounded by the indifferent mesenchyme cells. The clefts and spaces enlarge, fuse together to shape the blood-vessels while the lining mesenchyme cells flatten and form the endothelium. The microscopical specimens of Moshkowitz lend great force to this theory. He ascribes to blood-vessels the important part in the process of ossification in bringing a dead or inert tissue into heteroplastic activity; and in describing further how fibroblasts later become endothelium he shows that normal and pathological ossification are based upon identical features.

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[43] Koga—Kyota Igaku Zassi. Bd. IX, 1912, h. 2 and 3.

[44] Op. citat.

[45] The Relation of Angiogenesis to Ossification. Based upon the study of five cases of Calcification and Ossification of the Ovary. Johns Hopkins Hospital Bulletin, No. 301, March, 1916.

[46] *American Journal of the Medic. Sciences,* No. 531, p. 868; June, 1916.

In his conclusions, he reiterates " the importance of circumscribed lime deposits for the formation of primary Haversian canals, accomplished by the genesis of an active mesoblastic tissue, both around the surface and within the interior of such deposits. This mesoblastic tissue is derived from the adjacent blood-vessels and the predominant activity in the development of new blood-vessels." . . . .

The development of new blood-vessels affords the keynote to the interpretation in terms of cellular ontogeny of the process of ossification. The histological constituents which enter into the formation of new blood-vessels are the progenitors of all the histological compounds of osseous tissue. In other words, blood-vessels, osteoblasts, bone cells and marrow (in large part at least) are merely differentiations of the mesenchymal cell-unit. After having demonstrated that his specimens furnish strong corroboration of the adaptive or mesenchymal theory of angiogenesis, and to the theory of the non-specificity of endothelium, he adds that ossification does not occur without preliminary calcification, and calcification occurs only in dead tissues. We quoted Moshkowitz more extensively, as our own specimens show many resemblances to his, after calcification has occurred. Outerbridge gives virtually identical pictures but does not enter into their interpretations, but simply chronicles the calcification and final ossification.

Although Haga and Fujimura[47] do not ascribe a basic importance to the primary hemorrhage in the traumatic myositis ossificans, the reports of their microscopical findings in extirpated specimens of their cases variously mention in every one the presence of blood-extravasations; as " capillaries in the intermuscular connective tissue are torn and extravasation of blood has taken place . . . numerous and large hemorrhagic extravasation and mighty development of new capillary vessels . . . goodly sized blood extravasation in the intramuscular connective tissue . . . here and there blood extravasates and neoformation of capillaries . . :" and thus permit a different view of their importance from that of the authors.

Van Arsdale[48] published a very interesting and important case of an ossifying hæmatoma, important for this question of participation of free blood in the act of ossification. A young man, 22 years old, received about the middle of April, 1891, severe blows on his right arm. The region immediately below the insertion of the deltoid was the seat of a large, soft fluctuating tumor, showing an increase of circumference over its fellow of 10 cm. The skin over the swelling was bluish and the parts tender on pressure. When seen again, May 20, the circumference of the right arm was diminished 6 cms., tumor of bony hardness throughout and apices harder than middle. Presented on May 27 to Surgical Society of New York; diagnosis, ossifying hæmatoma. Operation June 22. External tumor 9 cm. and 3 cm. broad or high, external coat or shell nearly 1½ cm. thick; contains cavity filled with partly coagulated blood. Bony hardness of the wall most marked over periphery of tumor. Induration then gradually decreased toward central cavity.

The same course, from calcification to ossification, as seen in humans, was

[47] Arch. f. kl. Chir. Bd. 72, Hft. 1.

[48] ANNALS OF SURGERY, 1893.

observed in the one case published as such from animal pathology. Lorge[*] dissected an anatomy horse which showed the symptoms of progressive myositis ossificans. The horse showed in the connective tissue surrounding the smaller muscular bundles (perimysium internum) a great number of fine needles resembling cement, which made a noise when the knife passed over them. The affected muscular fibres had a pale yellowish color; they were atrophic and their contents partly fattily degenerated.

The diseased muscles were distributed all over from head to feet. In the tongue the mass could be kneaded with the fingers; in other places it was so firm that it could be crushed only with difficulty. Microscopical examination showed that the normal striation of the diseased muscles had disappeared and in their place were granular or dark dots composed of bone cells. The external cellular sheath of the muscle did not show anything of a pathological calcification. According to the views of Lorge, "the calcareous deposit in the inner sheath of the muscle was the first, the atrophy and fatty degeneration of the muscle fibre the second stage of a disease, which is rarely observed with humans and has been named progressive ossifying myositis by Professor Dusch, of Heidelberg."

Another animal case not yet published is one observed by Prof. Dr. K. F. Meyer, of the Hooper Foundation for Medical Research of Medical School, University of California. While showing my slides to the Doctor he was reminded of the case of a dog examined by him in Philadelphia in 1912 as Director of the Pathological Laboratory, Pennsylvania State Live Stock Sanitary Board, University of Pennsylvania. From comparing our notes and pathological findings we both came to the conclusion that this dog, the exact diagnosis of whose disease had been rather baffling, had been suffering from myositis ossificans progressiva, and Doctor Meyer was kind enough to permit me to incorporate his hitherto unpublished manuscript in my treatise. I give it largely in its original form, with a few changes made by me relative to its new viewpoint and submitted to and approved by Doctor Meyer.

In November, 1912, through the courtesy of Doctors Cunningham, Hoskins and Jarrett, of the Pennsylvania State Live Stock Sanitary Board, a dead setter dog was sent into the Pathological Laboratory which showed on all four legs a most remarkable anomaly which had developed gradually. Unfortunately, the dog had died the day previous, and therefore the carcass when brought to autopsy was in a fairly advanced stage of decomposition. Before giving the autopsy findings, I will briefly relate the clinical history given to me by the owner of the dog (Doctor Cunningham):

Setter dog; male; was born in May, 1909, and was one of a litter of fourteen. Some of the pups were killed accidentally by the mother. Seven were raised. They were all extremely nervous. At the age of four to five months the pup in question was lame in the left hind leg. It was found that the left hock region was swollen and on palpation very tender. The animal was treated with Fowler's solution for about one month, at the end of which time the swelling disappeared and the dog developed normally to a typical English setter and at the age of one year won two first prizes. At the age of eighteen months the dog became listless, would lie in the corner and refused to play, which was strange, as the dog had always shown unusual vivacity. At the age of nineteen months the swelling on the hock again appeared, was tender on palpation and soon extended above and below the hock. The dog became extremely lame and was most of the time dragging his legs under him. It was again treated with Fowler's solution, and about two months later a similar swelling started in the right hock, and at the age of twenty-two months both hind legs were enormously enlarged. A careful examination revealed that the joints were absolutely

[*] Annales de Médecine vétérinaire; publ. a Bruxelles XXme anneé; Mars.-Mai, 1871, ref. Repertorium d. Thirhlkde, Hering.

normal. The general condition of health was perfect; the appetite was good; no fever was ever noticed.

In the spring of 1912 a similar swelling started gradually, beginning on the metacarpus and elbow on the front legs. The dog started a very troublesome cough. This condition remained the same until November, 1912. He was able to walk by dragging his legs after him, but he had always to be assisted when walking up or down stairs. Two days before his death the cough was very bad and temperatures from 103 to 106 were noted. He died with the symptoms of heart paralysis November 19, 1912.

The autopsy was performed by me on November 20, 1912, and the following is a condensed report of the essential findings only:

Setter dog; male; 2½ years old. Emaciation; all four legs hard, tremendously enlarged and heavy. Fat tissue has nearly disappeared. Muscles on extremities atrophic, pale and ossified. Along the extremities the skin can only be removed with difficulty on account of a heavy layer of dense, hard, whitish connective tissue. Along the shoulder-blade at the insertion of the muscles, hard, nodular, fibrous, callous-like connective-tissue layers.

In both pleural cavities there is a small amount of reddish fluid. Costal pleura slightly reddish.

The right apical lobe is attached to the costal pleura by several broad connective-tissue bridges. All lobes of the lungs are œdematous and soft. The right upper lobe shows a tumor size 15½x19½ cm. distinctly protruding above the surface of the healthy lung tissue. Its color is whitish and in certain places grayish.

The consistency of this tumor is dense and hard. On section there is a connective-tissue capsule of about 6 mm., a whitish, yellowish tissue meshwork, which contains many cheesy, pus-like areas. The bronchi leading to this portion contain granular pus and a few superficial ulcerations. All the other lobes of the lungs contain in the bronchi a serous reddish fluid. The tissue is soft and friable.

The left and right bronchial lymph-nodes are enlarged and contain numerous yellowish nodules, which are in places also cheesy. In the trachea and bronchi a small amount of granular mucus intermixed with pus flakes. The vocal cords and the epiglottis are somewhat thickened and hard.

An examination of the extremities revealed the following conditions: The left and right thoracic limbs show the length of the ulna about 22 cm., a circumference of 22½ cm. and a thickness of 6 cm. The elbow and carpal joint are immobile. The metacarpal and interphalangeal joints are, under restriction, mobile. On incision the enlarged parts of the ulna show a fibrous, nodular, gritty, bone-like material. Both pelvic limbs are extremely enlarged, but remarkably flattened. The following figures will indicate the conditions:

The length of the right tibia is 25 cm. From the middle of tarsus to the end of the third toe, 18 cm. The diameter of the oval-shaped tibial circumference is 8 cm., and 6 cm. thick, and is considerably flattened in the middle. The circumference is 21.5 cm. The circumference of the metatarsal bones is 25 cm., and the diameter of the region of the tarsal bones is 10 cm. The second toe has a length of 3 cm. and a circumference of 5 cm., and is extremely plump. The structure of the subcutis is the same as that described for the fore-limbs.

No swellings or enlargements of any kind were noticed on the head, around the spinal column or the tail.

The left thoracic and the right pelvic limbs, together with the entire skeleton, were carefully macerated. The right thoracic and left pelvic limbs were fixed in Mueller's solution. The tumor in the apical lobe was fixed in formalin.

The maceration of the skeleton revealed the following pathological changes:

The supraoccipital and the condyloid fossa show slight roughening. The condyles

of the mandible show small, wart-like roughenings. All the bones of the head are somewhat porous.

The left shoulder-blade is light when compared with those of normal dogs; somewhat porous, but normal in shape. The posterior angle shows plump, thick, hard swelling, size 2½ x 4 cm. (See plate 1, 2, 3.) The edges are nodular, wart-like. The acromion is enlarged, flattened and porous. The scapular notch shows small, wart-like, spongy exostoses. The scapulo-humeral joint is absolutely smooth and normal. The humerus is normal in length, but extremely plump; shows on the crest a flat, leaf-like growth of bone tissue, which extends over the shaft, leaving a space free for the atrophic biceps muscle. Both the external and the internal condyloid crest, the external and internal condyle, the olecranon fossa, are covered by heavy (3 cm.), thick, spongy, wart-like osteophytes, resembling on the internal condyle the picture of a sponge. Both articular ends of the humerus are normal. The bone tissue is extremely spongy and soft. Above the condyles is a small hole connecting the coronoid fossa with the olecranon fossa and is caused by osteophytes on the proc. anconeus of the ulna.

Both ulna and tibia are covered with most luxuriant osteophytes. Their length appears about normal as compared with dogs of the same age. The existence of the ulna can only be determined on the proximal joint ends, where the radius is still separated from the ulna by a small groove. Otherwise the entire ulna is buried in the mass of new bone tissue. The thickness of this neoformation examined from the anterior side is about 5½ cm.; spongy, irregular masses of bone tissue surround the entire radius, leaving only on the internal side a round groove for the localization of the deep flexor and the flexor carpi internus. The joint ends are entirely invisible when mounted, but show distinct pseudo-arthrosis of the osteophytes. A similar pseudo-arthrosis is seen at the proximal end of the osteophytic growth and the osteophyte above the coronoid fossa.

The carpal bones are invisible on account of the masses of osteophytes. On the posterior view they can still with some difficulty be distinguished, as there the osteophytes have left a flat, oval channel for the passage of the flexor tendons. The metacarpal bones are uniformly covered in the anterior portion by an arborescent wart-like growth of osteophytes. The third and fourth bones are separated by a cleft. From the fifth and the first metacarpal bones the osteophytes grow over the posterior part of the metacarpus, forming the roof of the channel mentioned above. The osteophytes on the metacarpal bones show pseudo-arthrosis with the osteophytes of the carpal bones, the ulna and the radius. We have two masses of osteophytes, therefore, those of the radius and ulna and those of the carpal and metacarpal bones, which are connected by joints.

The first phalanges are plump; those of the fifth, second and first digits show the osteophytes on the external side. The second phalanx of all digits is chunky and shows wart-like osteophytes growing on the back of the phalanx. The wing and coronary ridge on the third phalanx are also plump and show crest-like osteophytes surrounding the entire bone. Only the ungual surface is entirely smooth, somewhat porous. All of the joint surfaces are smooth and unaffected. The volar sesamoids were lost during the process of maceration.

The pelvis appears normal, is somewhat porous; around the psoas tubercle and the iliopectineal line there are several wart-like osteophytes.

The femur is of normal length; shows similar conditions to the humerus; the proximal and distal joint-ends are normal.

The patella and the sesamoid bones are covered with osteophytes which are connected by pseudo-articulations.

The tibia and fibula are remarkably enlarged and show most of the lesions that were observed in the thoracic limb. Here also a space is left open for the flexor tendons, the lesions on the tarsus and metatarsus and digits are nearly identical with those just described for the thoracic limb, with the exception that there is a marked

lateral compression, probably due to the fact that the dog was lying constantly on the hind legs.

Most of the lesions are therefore seen on both extremities. They are characterized by extremely massive apposition of bone which is located around the compact masses of the bones, selecting frequently the places of the insertion of the tendons.

A chemical analysis of some of the osteophytes on the radius, and, for comparison, from the shoulder-blade, was made. I am indebted for the analysis to Dr. John Marshall, of the University of Pennsylvania.

CHEMICAL COMPOSITION OF DRIED BONE

	Normal	Exostosis	Shoulder-blade
Calcium phosphate, $Ca_3(PO_4)_2$	57.35%	53.98%	52.71%
Magnesium phosphate $Mg_3(PO_4)_2$	2.05%	2.75%	2.18%
Carbonate, fluoride, and chloride of calcium	3.45%	3.04%	3.32%
Organic matter (ostein)	33.30%	31.91%	32.58%
Water at 105°–120° C	.....	8.32%	9.21%

By the above table it is shown that the minerals are uniformly reduced as well as the ostein. A slight increase of water is noted and conditions are prevailing here which resemble somewhat those found in osteomalacia.

The microscopical examination of sections of the lung tumor, stained with hæmatoxylin, eosin, Van Gieson, etc., show:

A thick layer of connective tissue containing a few capillaries. Below this layer is seen the tumor tissue of alveolar or papillomatous epithelium. Connective tissue septa of varying thicknesses form a net-like stratum; the free spaces, tubules or alveolæ are coated by single or stratified layers of fairly regular epithelial cells and contain débris, desquamated cells and coagulated masses.

The size of the alveolæ is varying; they are mostly tubule-like; in some places they are glandular and resemble adenomatous or papillomatous structures. The epithelial cells are squamous, with large vesicular nuclei; sometimes they are narrow and cylindrical; in most alveolæ they are in single layers. Most of the cells have smooth contours; some, however, show filament-like protoplasmatic projections. The type is apparently one of metaplastic pulmonary epithelium. It is impossible to determine whether alveolar epithelium of the lung, or whether metaplastic columnar epithelium of the bronchial lining is the substratum from which these tumor cells have developed. In specially stained sections no mucin-like material could be seen, so that I feel justified in excluding the possibility of the tumor cells having originated from the mucous bronchial glands.

In the depth of the tumor the tissues are to a large degree necrotic and consist of material which stains deeply with eosin; some areas of liquefaction are distinctly seen as indicated by the presence of numerous leucocytes and lymphocytes.

The lymph-nodes showed the same papillomatous arrangement of the same metaplastic epithelial cells. A careful search for tubercular lesions was negative. The lung tumor can therefore be diagnosed as a primary lung carcinoma with secondary metamorphosis and metastases in the bronchial lymph-nodes. It was impossible to determine if the tumor was of *alveolar* or of *bronchial origin*.

Sections of the bone proliferations which were fixed in Mueller's solution and decalcified with nitric acid and embedded in celloidin show on the left fibula (for example) that the entire bone material directly under the derma is covered by fibrous connective-tissue layers. The osteophytes in form of lamellæ and trabecula are rich in blood-vessels and Haversian canals. Some of the walls of the blood-vessels are abnormally thick. Fibrils of connective tissues cross the muscles in different directions and compress the fibres. Between the osteophytes is a soft, succulent connective-tissue mass.

The compact substance of the fibula is extremely reduced in thickness—and it is difficult to realize what is originally bone and what is osteophyte. The Haversian canals are very remarkably enlarged; one notices groups of osteoclasts and giant cells scattered irregularly. The process here is of the usual character of bone resorption.

Below this zone similar trabecules grow into the marrow. The structure and shape are similar to the periosteal osteophytes. Dense connective-tissue fibres show layers of osteoid tissue which is surrounded by spindle-shaped cells resembling osteoblasts. The process of active bone apposition is quite apparent. Lack of comparative material prevented a further study of this enchondral ossification.

In view of a recent revision of the data of this case I do not doubt it to be one of myositis ossificans progressiva in the dog. The clinical history is an exact counterpart of many of the typical histories of human cases in the foregoing compilation (*vide* cases 15, 17, 23, 25 and many others), the more extended ossification having been arrested by an early death due to complication with the carcinoma in the lung. The pathological changes in the osseous system are significant of the disease. The ossification along the muscular tracks and insertions involving and supplanting these structures, the comparative freedom of the articulations, together with the spongious character of the new bone formation, are all features of this disease. A very characteristic symptom marking this case undeniably as one of myositis ossificans progressiva is the microdactylic state of the thumb of the photographed thoracic extremity. The end phalanx was lost in the process of maceration, but the first metacarpal is less than half of the normal size and the first phalanx also is considerably shortened. At the time of the autopsy the diagnosis of myositis ossificans progressiva was not taken into consideration and therefore no attention was paid to the fingers and toes of the other extremities; but the photograph of the macerated foot shows the condition distinctly so as to remove all doubt, at least as to this extremity. The very rare similar observations in the literature of animal pathology appear from this viewpoint in a new light.

In 1890, Kitt[80] observed a case which, according to his description, resembles my specimens in many respects. The exception was the exceedingly prominent lesions on the mandible very frequent in human cases of myositis ossificans progressiva. Perhaps inasmuch as the dog had been destroyed in perfect health, the lesions on the limbs could not develop to the same degree as those seen in the case under discussion. The dog was one year old. Kitt[81] explained the condition as an abnormal diathesis of bone formation, and inasmuch as the ossification started at the insertion of the muscles, he diagnosed his case as a multiple myopathic hyperplasia of the bones.

Liénaux[82] saw two old dogs with distinct enlargement of the thoracic and pelvic limbs. He could autopsy, however, one animal only. The pictures and descriptions correspond with my observations only that the extent of the process is a less marked one than in my case. In another case, of which he found only the skeleton in the

[80] Kitt: Lehrbuch der pathologischen Anatomie der Haustiere, I, 1910, 4th edit., p. 388.

[81] Symmetrische multiple Hyperostosis & Exostosis bei einem Hunde tahresbericht der kgl. tierarztlichen Hochschule, 1890.

[82] Liénaux: Apropos de l'ostéitisme, ostéite déformante du chien. Ann. de médecine vétérinaire. Mars, 1899.

FIG. 27.—Apparent erythrocyte participation in bone formation and differentiation of osseous and cartilaginous parts of ring. *a*, Erythrocytes; *b*, cartilaginous part of ring; *c*, osseous part.

A *B*

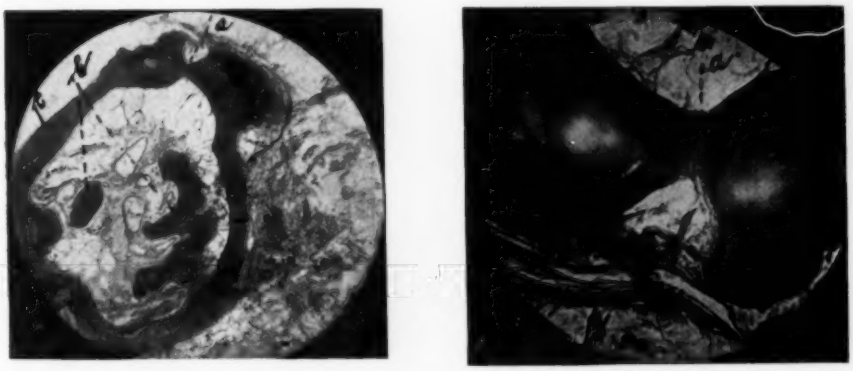
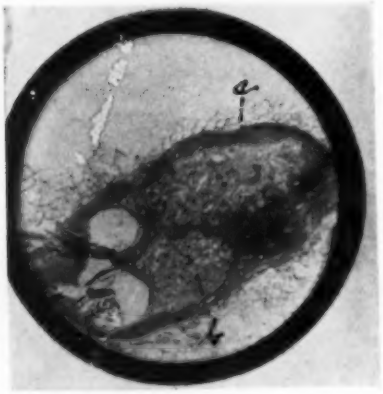
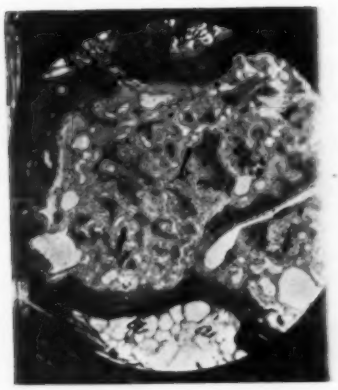


FIG. 28.—Interior osseous branches and islets forming and a connective-tissue bridge between bony ends of ring. *A*, *a*, connective-tissue bridge; *b*, branches and islets; *c*, outer ring (oc. 6 obj. 16). *B*, *a*, connective-tissue bridge of *A* (oc. 6, obj. 4).

A



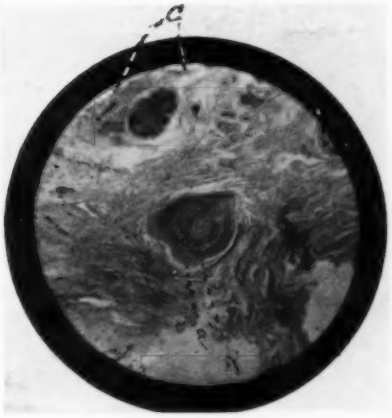
B



C

FIG. 29.—Bone and bone-marrow. *A*. *a*, outer ring. *b*, bone marrow (oc. 2, obj. a'7). *B*, *a*, outer ring; *b*, bone-marrow (oc. 2, obj. 16). *C*, *a*, branches; *b*; fibroblasts and capillaries (oc. 2, obj. 4). (Enlargement of inner part of *A*.)

A



B

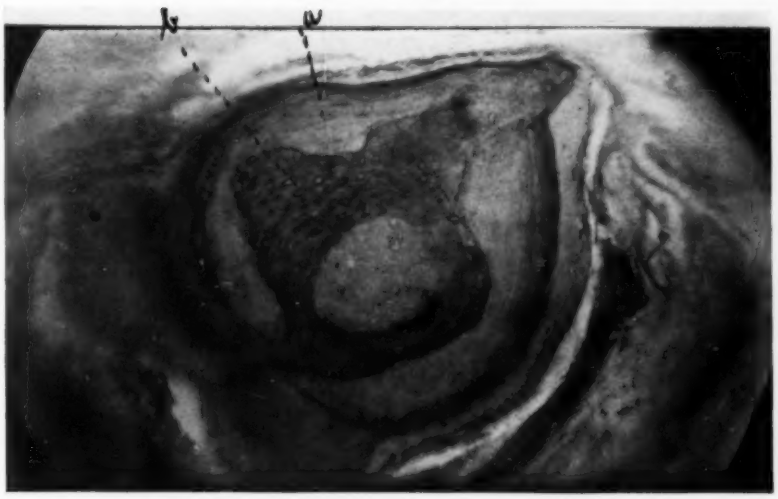
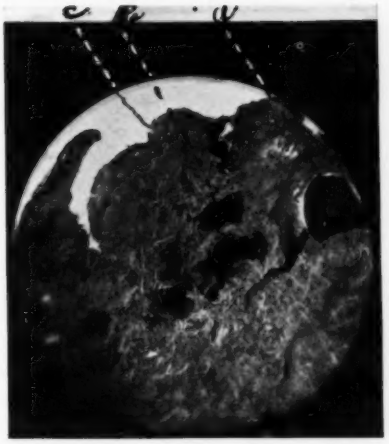


FIG. 30.—Cartilaginous development of bone. Cartilaginous tissue at inner border of osseous ring. *A*. *a*, ossified outer part; *b*, cartilaginous part; *c*, epidermis (oc. 2, obj. 16). *B*. *a*, ossified part; *b*, cartilaginous part (oc. 2, obj. 8). (Enlargement of *A*.)

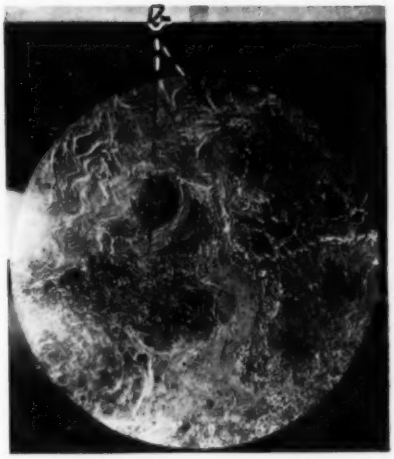
A

B

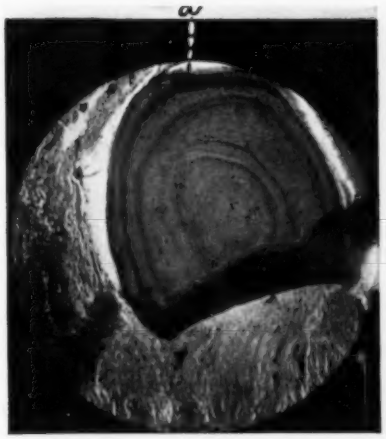
FIG. 31.—Inner branches with erythrocyte participation. *A*, *a*, erythrocytes entering in bone formation (oc. 6, obj. 16). *B*, *a*, erythrocytes in same place (oc. 6, obj. 4). *B*. Branches of later stage of formation with apparent erythrocyte participation (enlargement of *A*). *a*, erythrocytes; *a'*, erythrocytes in participation of building of branches; *b*, branches.



A

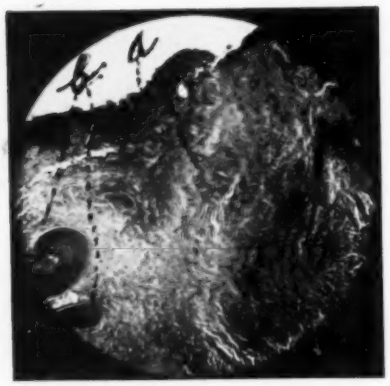


B

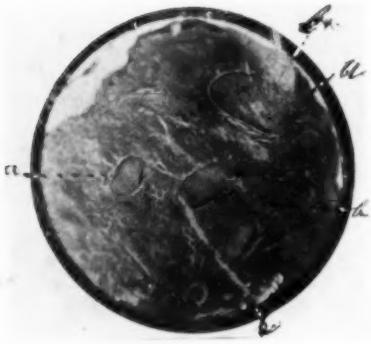


C

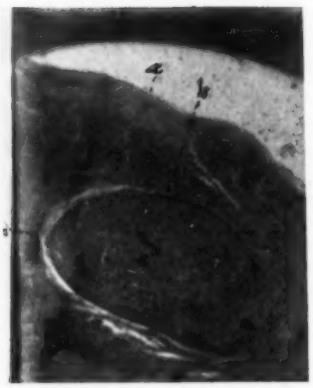
FIG. 32.—Solidified bone and primary hemorrhage both near each other close to epidermis. *A*. *a*, solidified bone; *b*, primary hemorrhage; *c*, erpidermis (oc. 2, obj. 2 *). *B*, *b*, primary hemorrhage (oc. 6, obj. 16). *C*. *a*, solidified bone (oc. 6, obj. 16).



A



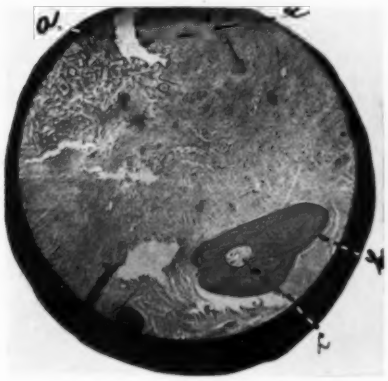
B



C

FIG. 33.—Partially solidified bone with central remainder of erythrocytes close to epidermis. *A*, *a*, epidermis; *b*, bone with erythrocyte centre (oc. 2, obj. 2 *). *B*, *a*, epidermis; *b*, bone with erythrocyte remainder (oc. 2, obj. 2 *). *C*, *a*, epidermis; *b*, same bone; *c*, erythrocyte remains (oc. 2, obj. 4).

A



B

C

FIG. 34.—Solidifying bone with remainder of erythrocyte central enclosure. *A*, *a*, epidermis; *b*, bone; *c*, erythrocyte central enclosures (oc. 2, obj. a *). *B*, *b*, bone; *c*, erythrocyte central enclosures (oc. 2, obj. 16). *C*, *a*, erythrocyte remainders; *b*, erythrocytes (oc. 6, obj. 4).

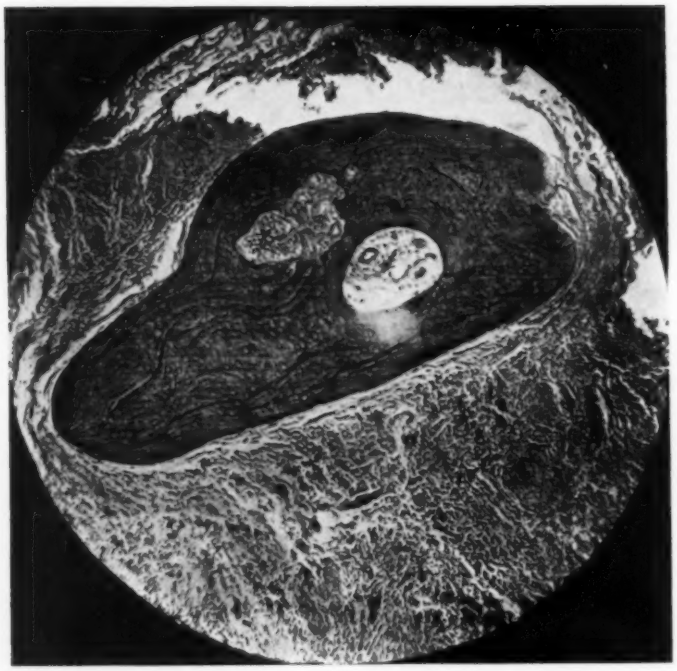


FIG. 34.—*D*. Bone in late stage of solidification with small remainders of hemorrhagic enclosure. *a*, outer bone layers; *b*, remainder of hemorrhagic enclosure. (Enlargement of Fig. 34 *C*.)

museum at Brussels, were seen similar symmetrical exostoses. These cases were diagnosed by him as ostitis deformans. Ten years later, on account of some further observation along the same subject and influenced by the work of Alamartine[83], the same writer revised his early conception of the bone lesion and considered it to be identical with the so-called "osteo-arthopathia hypertrophiante" in man. Ball and Alamartine[84] had observed a tubercular dog which had the same macroscopic and microscopic processes as those seen by Liénaux, and therefore it suggested itself to Liénaux[85] that the term "osteoarthropathy" would be better than "diffuse osteo-periostitis" or "ostitis deformans."

The few cases described by Kitt and Liénaux show great analogies with my case. The processes, however, are not as far advanced in either case, and there are also differences in the ages of the dogs affected. The animal seen by Kitt was one year old; those reported on by Liénaux were all over ten years old when first seen. Aside from these minor differences, which also occur frequently in human patients, the cases are identical with mine so far as concerns the anatomical lesions of the bone system.

In none of these cases a satisfactory clinical or causal explanation has been given; the tuberculosis in Ball and Alamartine's case was an accidental complication, and they all belong most probably to and should be classified as myositis ossificans progressiva of the dog.

In order to arrive at a better understanding of the basic features of our malady, it appears advisable to me, before we enter into a discussion of the peculiar and specific characteristics of the progressive ossifying myositis, that we should now consider collectively all those traits it possesses in common with its near relative, the traumatic myositis ossificans and its experimental imitation. They all center around the pathologic anatomical structural pictures and bone-forming process; they are identical in appearance and modes of procedure in both affections.

In fixing its place among the pathological entities it occurs at once that Virchow's view of its being a multiple exostosis must be abandoned in spite of the master's word, and the free growth conceded within the confines of the muscle and its surrounding connective tissue.

We have the research work of many authors, including our own case, to establish the evidence of independent growth of bone tissue within the muscle bundles and their connective-tissue envelopings and dividing partitions.

That question also as to the primary point of attack, still indicated by the name myositis, may be considered settled. Every observer who has excised a piece of the affected tissue at an early stage of the local pathological change has found the inter- and intramuscular connective tissue to be the primary

[83] Alamartine: Ostéo-arthropathies hypertrophiantes d'origine tuberculeuse. Revue de chirurgie 35, 1907, p. 992.

[84] Ball & Alamartine: Pathologie & Ostéo-arthropathie hypertrophiante d'origine tuberculeuse chez l'homme et chez le chien. Revue de chirurgie, 1908.

[85] Liénaux: Remarque au sujet de la nature de l'ostéo-arthropathie hypertrophiante. Bull. de l'Académie Royale de Médecine de Belgique. 27 février, 1909.

seat of attack, even before my discovery of isolated bone-tissue in the skin removed the possibility of doubt. In both diseases the muscle fibres are the secondary sufferers and succumb only to the increasing pressure and embodying force of the growing ossification. While these two questions are easily absolved, we encounter a greater difficulty in order to arrive at a satisfactory result in establishing the manner and the identity of the bone-forming process.

Although the oldest attempt to explain the heterotopic ossification by a metabolic retention of lime has been refuted by the exact modern investigations of both Burgerhout[56] and Austin[57], the fact that its presence as a local deposit is an essential condition for bone-formation continues to be valid. The truth of this assertion has been proven by the experimental work of Dibbelt[58] through calcium supply by way of the alimentary canal, and by hypodermic injection of 1 per cent. solution of chloride of calcium of Koga[59], this latter successfully repeated by Goto[60]. Many other authors confirm it by their observations on humans and point at the initial hemorrhage as the source of the calcium supply in spite of its rather low percentage in the blood. The idea of looking to the blood as the first cause of ossification, either as a direct irritant for its initiative stages or as a chemical one through its lime deposit, lies very close if one considers that in cases of traumatic ossifying myositis the first effect of the trauma is always a hemorrhage into the injured tissues.

We may dispense in these cases with a serious consideration of the periosteal participation, as only the entirely severed and intramuscularly implanted periosteum gives rise to a free bone-formation, whilst the flap always shows the new-formed bone in connection with the underlying skeletal bone, and it would be more than unreasonable to presume that the great majority of traumatisms responsible for a case of skeletal-free myositis ossificans tear off a piece of periosteum and transplant it into the adjoining muscular tissue.

Many clinicians, as we have seen, were able to witness the growth of the newly-formed bone directly from the initial hemorrhage to the final extirpation of the finished product. The well-known Schede blood-clot for the filling of bone cavities has often served its purpose and the microscopical pictures of early stages of traumatic as well as progressive cases show so constantly the more or less well-preserved remnants of an infiltrating blood-extravasation close to or surrounding the various stages of ossification, from earliest connective-tissue hypertrophy and calcification to complete ossification, that the causative connection becomes irresistibly impressive. Such an impression, gradually rising to a firm conviction, I have also

[56] Op. cit.
[57] Op. cit.
[58] Op. cit.
[59] Op. cit.
[60] Op. cit.

gained from the study of my slides, and in their description and illustration I have tried to convey this conviction to my readers. The various anatomical regions show the progress of bone-formation on different paths; osteoblasts with a fibrous or cellular cartilaginous route in the intra- and intermuscular connective tissue to direct calcification and osseous growth in the hypertrophic fibrous connective tissue of the skin, all starting from the primary blood clot. But with even the fullest admission of the blood's causative ossifying action, we have moved but one step farther toward the solution of the mysterious problem. The question arises now, what are the determining conditions to induce in some cases an ossification after a subcutaneous tissue-invading hemorrhage, and in the vast majority of other similar traumatic cases simply an uninteresting and uninterrupted absorption?

In the description of my microscopical pictures I drew attention to a tissue zone surrounding a developing ossifying part, which zone refused to take the stain well, and with its peculiarly indistinct cell and tissue differentiation showed the unmistakable picture of tissue-necrosis.

A similar area, although not specially mentioned as such, may be seen in Goto's microphotographs of his experimental myositis ossificans produced by means of the chloride of calcium injections.

This tissue-necrosis, following the initial hemorrhage, we believe to be another determining factor for the comparatively extremely rare sequel of ossification to muscular traumatism. This tissue-necrosis, as a preliminary condition to calcification and later ossification, is mentioned by Rohmer[61] in his description of bone-formation in focal endocarditic and endarteritic calcareous necrotic ulceration, of which latter Moenckeberg[62] met with ten in 100 cases of senile arteriosclerosis. Pollak[63] found that the ossification took place in all his cases around calcified necrotic foci in the lungs; and let us recall here that local calcifications of necrotic caseous foci are not infrequently seen in old ulcerative pulmonary processes.

Sacerdotti and Frattin[64] produced ossification in secondary necrotic foci of the kidneys, the blood-vessels of which they had ligated, and thereby caused primary hemorrhages. Liek[65] obtained identical results.

J. F. Poscharissky[66] examined heteroplastic osteogenesis in lungs, heart, blood-vessels, lymphatic glands, brain, eye, uterus, liver, thyroid gland, and always met with regressive metamorphosis (necrotic foci) as the primary incentive for calcification which was followed by ossification. He found that there exists a kind of an antagonism between the necrotic centre and the living periphery of the affected tissue nodule, which is being demonstrated by an effort of the connective tissue to enter into the necrotic centre,

[61] Op. citat.

[62] Ueber Knochenbildgn. in d. Arterienwand. Virch. Arch., Bd. 167, 1902.

[63] Op. citat.

[64] Op. citat.

[65] Experimenteller Beitrag. z. Frage d. heterogenen. Knochenbld. Arch. f. kl. Chirurg., Bd. 80, p. 279.

[66] Beitraege (Ziegler's) z. pathol. Anat. & allgem. Pathologie, Bd. xxxviii, p. 135.

shows a constant tendency to grow. He concludes that osteogenesis may occur in any organ of the human body if there exists a necrosis of the tissue, or at least a sclerosis so intense that it is interfering with the natural blood supply and accompanied by an incrustation with calcium salts. In all his observations Poscharissky never encountered the formation of cartilaginous tissue.

A. Barth shows, in his experiments [67] on bone implantation, new growth of bone only took place after a soil of dead tissue had been provided for it and that[68] calcium favors rapid ossification.

S. Ehrlich[69] holds that the iron when available (from hemorrhages) serves as a mordant for the calcium which is subsequently deposited.

H. Gideon Wells[70] finds as a result of his own and others' experiments that calcification, as the preliminary stage of pathologic ossification, takes place only in dead (necrotic) tissue or in such of low vitality and insufficient circulation. Physiologically, calcification in old age takes place also in ill-nourished tissue, like the laryngeal or costal cartilages.

He gives the amount of calcium contained in blood (serum) of mammals as from 0.11 to 0.131 per cent.,[71] about two to four times as much as is soluble in water.

It may be seen that his conclusion is well supported by pathologic-anatomical evidence. The decision which of the two, the hemorrhage as calcium supplier, or the necrosis as the stimulating and receptive soil for calcification, is the more important for the final ossification, seems impossible to decide at present.

Goto's[72] experiments on rabbits and guinea pigs, in producing intramuscular hemorrhages by severe hammering of the quadriceps femoris never resulted in ossification; the very early excision after the traumatism showed calcifications of the torn muscular fibres amid the fresh hemorrhages; later excisions showed complete resorption of the hemorrhage and the destroyed tissue, in their stead simply a replacing connective-tissue hypertrophy. By the daily injections of 3 cm. of 1 per cent. chloride of lime solution into the quadriceps, continued for thirty consecutive days, bone-formation could be produced. A shorter period, however (eight days), only showed conditions similar to the early traumatic pictures. An unbiased interpretation of these experiments still leaves open the question of the more important factor, lime or necrosis. The lime supply alone by blood extravasation does not seem sufficient to induce the entire range leading to ossification if a reparative

[67] Beitraege (Ziegler's) z. pathol. Anatomie & allgem. Pathol. 1895, Bd. xvii, p. 65.

[68] Ueber kstl. Erzeugg. v. Knochgew. & ueber d. Ziele d. Osteoplastik. Berl. kl. W., 896, p. 8.

[69] Eisen & Kalkimpregnation in menschlichen Geweben. Centralbl. f. allg. Pathologie, 1906, xvii. p. 177.

[70] Calcification and Ossification. Archives of Internal Medicine, vol. vii, June, 1911, No. 6, p. 721.

[71] Op. cit.

[72] Op. cit.

process intervenes and resorption interrupts the progressive stages; is this reparation, however, prevented by a long continuance of lighter daily traumatisms assisted by the constant addition of calcium supply, both carried on by the intramuscular injections of comparatively large quantities of calcium chloride solution, then bone formation is induced.

The foregoing explanatory attempt, although valid for both the progressive as well as the traumatic ossifying myositis, only speaks for both *after* the occurrence of the initial hemorrhage.

While the cause of this extravasation needs no further explanation in the traumatic form of myositis ossificans, a hemorrhage as forerunner to ossification in the progressive form seems as inexplicable as its constant association with microdactylia as a characteristic symptom.

We have tried to lay the hemorrhage to a deficiency in coagulability of the patient's blood, but the tests we made did not bear us out; coagulation occurred even ahead of the normal time, and the reason for the precursory hemorrhage in myositis progressiva ossificans was not explained by any failure of proper coagulation.

It seems, moreover, apparent that all the strange characteristics of this disease are more or less interdependent and might be treated as due to a common cause. It may be possible to advance our understanding of these connective features by examining the progressive ossifying myositis in comparison with other maladies that offer certain symptoms common to both.

The feature of heredity in this disease has hitherto been treated as negligible, as in all former compilations only one case (No. 70, Faughan and Fanning) of hereditary transmission of disease and deformity had been ascertained. We have found two more instances, one (No. 30) and (No. 81) a case of Gaster, going through three successive generations, grandfather, father and three sons, with a selective preference for the male members of the families, who, besides being victims of the ossifications, showed, every one of them, the marked congenital finger and toe deformities.

This deformity, brachydactylia, occurring independently, has engaged the attention of many writers and aroused great interest on account of certain embryological phylogenetic questions. Brachydactylia may be due to either the shortness of a metacarpal or metatarsal bone, or of one or more phalanges, or of either combined, and it may be appropriate to differentiate them by calling the first brachydactylia, the second brachyphalangia and the third brachydactylphalangia or brachyphalangodactylia.

The two views, as to the causes entering into the production of carpal deformities, are the exogenetic, mechanical, and the endogenetic, embryological. The brilliant results of Tornier[73], in experimentally producing various deformities of hyperdactylia in tritons and other animals possessing similar regenerative force, lent great support to the mechanical explanation

[73] Ueber Hyperdaktylie Regeneration & Vererbung mit Experimenten. Arch. f. Entwicklgsmchk. d. Organismen. Bd. iii, 1896, p. 409.

of such deformities as hyper- and hypodactylia, syndactylia, etc., through the agency of amniotic bands and intra-uterine pressure.

Even for brachydactylia a similar explanation was advanced upon the theory of localized pressure from projections of uterine walls, although a clear conception how such pressure could act in the human as to influence the growth of a single metacarpal bone has never been given. Far less possible appears such a solution for symmetrical brachydactylia of one or more metacarpal bones, and we shall try and briefly give our reasons for rejecting this theory for brachydactylia, with which myositis ossificans progressiva is preëminently associated, and for claiming for this deformity an endogenetic cause.

We know that the "anlage" of the carpus consists of three parallel tissue rays (gewebstralen) which divide again in several parts of which the radial (or relatively tibial) and medial continue into the first two fingers (toes) and of the ulnar (fibular) ray which traces its origin secondarily from the ulna (fibula), issuing lateral branches from the fourth and fifth fingers, also for the basal elements of the third finger. Thus we see at first the "anlage" of first and second fingers, later of the third and last of the fourth and fifth. We find a certain relationship in the extent of deformities according to this normal mode of development.

The following references are only an approximately complete list of the literature of this deformity, and in order not to devote too much space to this section of my paper, the cases will be but briefly quoted:

M. Leboucq[14] mentions a case of bilateral symmetrical brachydactylia of all the fingers.

E. W. Roughton[15] demonstrated a case of brachydactylia of the right big toe in a 30-year-old man, and of the fourth toe on left foot. In the second case the brachydactylia affected the third, fourth and fifth toes of both feet.

Julius Sternberg[16] registered two cases; the first showed brachydactylia of both third fingers and both third toes; the second brachydactylia of both fifth toes.

E. E. Goldman[17] describes a case of brachydactylia of the third, fourth and fifth fingers of the right hand, and of the fourth and fifth fingers only on the left hand. The same patient showed symmetrical brachydactylia of both fourth and fifth toes.

L. Freund[18] reports a brachydactylia of both fourth fingers of a young man. The distal epiphyses were shortened and atrophied.

Rieder[19] mentions a man with an interesting case of combined brachydactylia of

[14] Brachydactylie et Hyperphalangie symétrique de l'Index et du Médicus Bulletin de l'académie Royale de Médecine de Belgique. Tome x, No. 5, 1896, p. 345. (This case had been presented already to the Med. Soc. of Ghent by Dr. M. Colson in 1883 and published in Pfitzner's essay on this subject.)

[15] Lancet, July 3, 1897.

[16] Symmetrische Verkuerzg. d. 3t. Mittelhdknoch. Wiener klin. Wschrft. 1902, No. 41, p. 1060.

[17] Beitrag. z. Lehr v. d. Missbldgn. d. Extrm. Beitraege z. klin. Chir., Bd. vii, 1891, p. 239.

[18] Die Brachydaktylie durch Metakarpalverkuerzung. Ztchrf. F. Heilkde. 1906, Abth. f. Chir. etc. Bd. xxvii, p. 129.

[19] Ueber gleichzeitiges Vorkommen von Brachy- und Hyperphalangie a. d. Hand. Deutsch. Arch. f. klin. Med., Bd. 66, 1899, p. 330.

the fourth finger of the right hand and hyperphalangia (three phalanges) of the thumb of the same hand. The daughter of this man had syndactylia of the right hand and hypophalangia of all other extremities.

Wagner[80] published the case of a woman who showed a bilateral brachydactylia of the third and a unilateral of the fourth fingers.

Hochheim[81] adds the case of a woman 18 years old with a bilateral brachydactylia of the third, fourth and fifth fingers and a brachydactylia of third and fourth toes, with shortened distal epiphysis and an imperfect development in breadth. A brother had a brachydactylia of the fourth right toe.

G. Joachimsthal[82] communicates two cases from his experience. He observed a woman of 27 years with a bilateral brachydactylia of the fourth and fifth and a brachyphalangia of the second and third fingers, and two women of 29 and 22 years, sisters of the former, with the same deformities, although not quite so pronounced. The only brother is normal, but has a daughter 1 year old with the same defects, and a son of the older sister had the identical deformities. In the same article were the very interesting cases of a young man with a bilateral brachyphalangia (middle phalanx) of the second and fifth fingers, whose mother was equally afflicted; a case of a girl 12 years old with a bilateral brachydactylia of the fifth finger, and of two women, 25 and 31 years old, each with bilateral brachydactylia of the fourth fingers.

From Cerechellis Institute, Carlo Fossetti[83] reports an observation of a bilateral brachydactylia of the fourth finger in a woman of 28 years. The distal epiphyses of the fourth metacarpals were shortened and atrophied.

Alfred Machol[84] describes the following cases from the Breslau clinic: Girl 17 years; brachydactylphalangia of the third finger of left hand. Besides a shortening of the third metacarpal bone, there existed also one of the basal phalanx. The distal epiphyses of the third metacarpal and the proximal one of the basal phalanx are rarefied and atrophied and demonstrate the cause of the deformity.

Woman of 34 years, shows a symmetrical brachydactylia of the fourth fingers and brachydactylia of the right fourth toe. The distal epiphysis of the fourth metacarpus is round like a ball and sits squarely on the diaphysis. The osseous structures of both metacarpal phalanges are rarefied.

A girl of 30 years with a unilateral brachydactylia of the fourth left toe. In this case also the osseous substance of the deformed toe is rarefied.

A girl of 12 years had scarlatina when two years old, followed by a paresis and debility of the entire right leg. Circumference over entire leg about 2 cm. less than the correspondingly right side. Brachydactylia of fourth toe of same side, osseous substance atrophied. Also smallness and atrophic state of phalangeal epiphysis, which may, however, be attributed to the generally impaired nutrition of that leg.

Boy 12 years old; poliomyelitis anterior when three years old. Pes varus sinister. Left foot slightly smaller than right. Brachydactylia of the fourth left toe. The osseous substance of left fourth metatarsus atrophic and bone-tissue rarefied. Distal epiphyses likewise compressed and squatty.

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[80] Beitrag zur Kenntniss der Brachydactylie. Fortschr a. d. Geb. d. Roentgensc. 1903, Bd. vii, pp. 94-98.

[81] Fall von Brachydaktylie. Fortschr. a. d. Geb. d. Roentgenstralen, 1904, Bd. vii, S. 273-76.

[82] Die angeborenen Verbildungen der oberen Extremitaeten. Ergaenzungsheft 2 d. Fortschritte auf dem Geb. d. Roentgenstralen. Hamburg, 1900.

[83] Brachidattilia simmetrica della mano. La clinica chirurgica 1914, No. 11, p. 239.

[84] Mitteilungen a. d. Grenzgebieten d. Medizin & Chir. (Mikulicz Gedenkband) Jena, 1907, p. 712.

Faulhaben[85] observed in a patient 22 years old symmetrical bilateral brachydactylia of the fourth and fifth fingers.

Fontana and Vachelli[86] encountered a most interesting triplicate of cases concerning two sisters, 16 and 12 years old, each with bilateral brachydactylia of the fourth fingers, and their mother, who showed this deformity on the left side only. Another woman, who did not belong to this family, also had a brachydactylia of the left fourth finger.

Lunghetti[87] relates the case of a man 32 years old with brachydactylia of both third fingers and brachyphalangia of the middle phalanx of the fifth fingers.

Kenyeres[88] saw a man of 37 years with symmetrical brachydactylia of the fourth fingers. The terminal phalanx of the left thumb was also about one-third shorter than that of the other side.

A. Mosenthal[89] describes (1) a brachydactylia of a young girl 20 years old affecting the fourth and fifth metacarpals of both hands (mother said to have had the same deformity), and showing a marked atrophy of these bones. (2) The cases of mother and daughter both with bilateral brachydactylia of the first metatarsus, with abduction of big toe (hallux varus).

Erich Ebsten[90] communicates in the same article an observation of brachydactylia of the left ring-finger and another case of a brachyphalangia of all four fingers of the left hand caused by the shortening of the middle phalanges only.

Similar cases of familiary heredity deformities of the fingers since the observation with the X-ray are but few, Julius Drey[91] had occasion to observe four generations of a family, the various members of which (thirteen normal, twelve defective) suffered from hypophalangia and digital synostosis.

Rich Pott[92] enjoyed a similar opportunity of observing in pre-X-ray times unmistakably hereditary defects in four successive generations. The members of this family, of which nine were normal, showed in five oblique nail phalanges of thumbs, three double end phalanges and three double entire thumbs (six complete fingers on each hand).

H. Drinkwater[93] reports two cases of hereditary transmission of digital deformities, members of both familes suffering from brachyphalangia. The deformity was caused in both families from an absence of the second phalangeal epiphysis, at the base only in one family, who showed in four generations twenty-six normal and twenty-one short-fingered members; the other in as many generations, with both epiphyses defective, had ten normal and nine short-fingered ones. This latter family was peculiarly interesting, as one of its normal married women had practiced illicit intercourse with a short-fingered man. She gave birth to one short-fingered son, who

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[85] Deutsche mediz. Wochenschr. 1904, p. 1454.

[86] Archiv. d. Ortoped. 1902.

[87] Sopre un caso brachydattilia simmetrica della mano. Archivio di Ortopedia Febr. 1902, p. 52.

[88] Fortschrite a. d. Geb. d. Roentgenstralen. Bd. ix, 1905-6, p. 351.

[89] Eleventh German Orthopædic Cong. Einige Faelle v. Brachydactylie. Verhdlgn. d. deutsch. Ges. f. orthop. Chir. 10th Congr. 1911, p. 36.

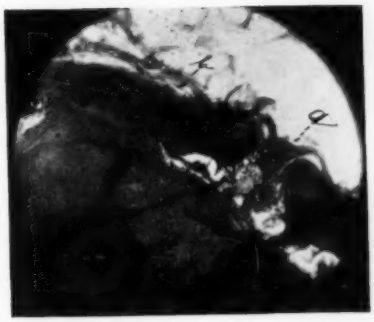
[90] Zur Aetiologie d. Brachydaktylie. Mitthelign. a. d. Grenzgebieten d. Mediz. und Chir. Bd. 21, 1910, p. 531.

[91] Hereditaere Brachydaktylie kombinirt mit Ankylose einzelner Fingergelenke. Festschrift gewidmet M. Kassowitz, p. 34, Berl., Julius Springen.

[92] Ein Beitrag. z. d. symmetr. Missbildgn. d. Finger & Zehen. Jhrbch. f. Kinderhlkde, Bd. xxi.

[93] Account of Family showing Minor Brachydactylia. Journal of Genetics, vol. ii, p. 21, 1912-13, p. 21.

A



B

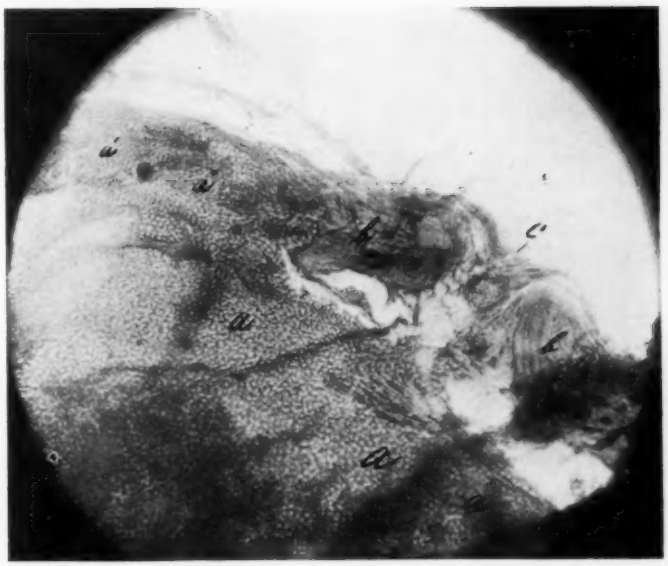


FIG. 35.—A, Connective-tissue bridge and predominant participation of erythrocyte in bone formation. *a*, connective-tissue bridge; *b*, erythrocytes participating preëminently on ring in bone formation (oc. 2, obj. 8). *B*. Connective-tissue bridge and predominant apparent erythrocyte participation in formation of ring (enlargement of Fig. *A*). *a*, erythrocytes; *a'*, erythrocytes in apparent participation of formation of ring; *b*, ring; *c*, connective-tissue bridge.

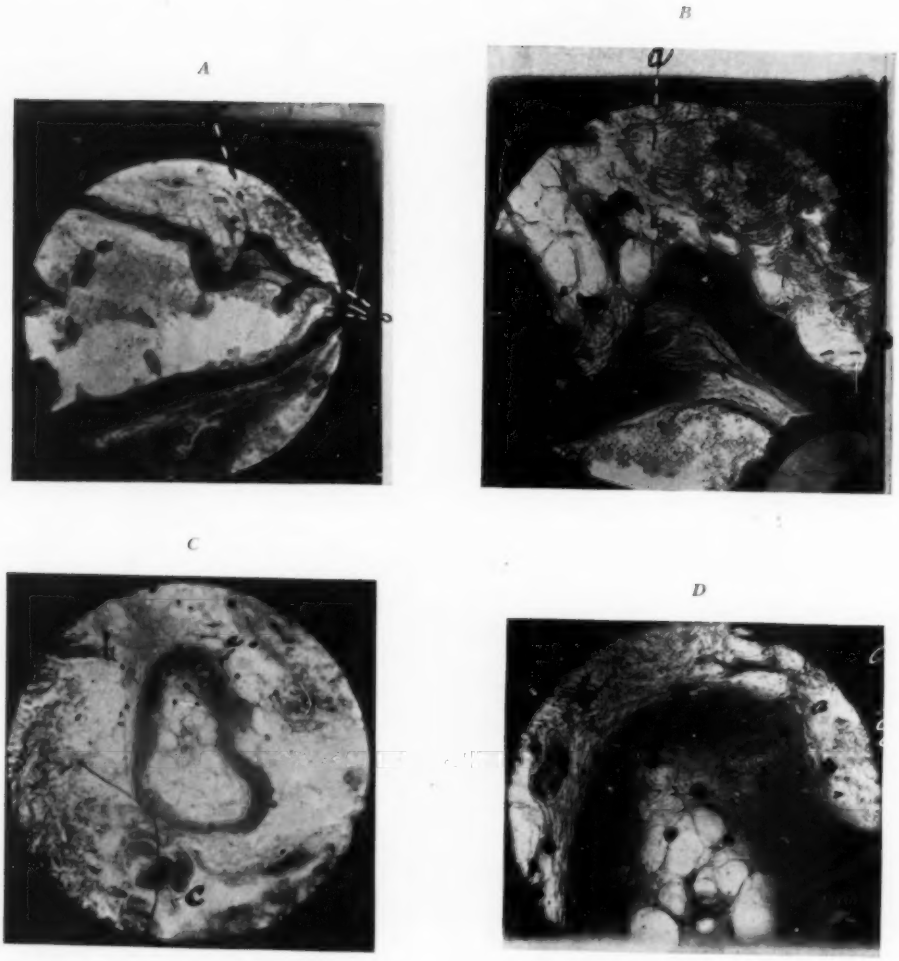


Fig. 36.

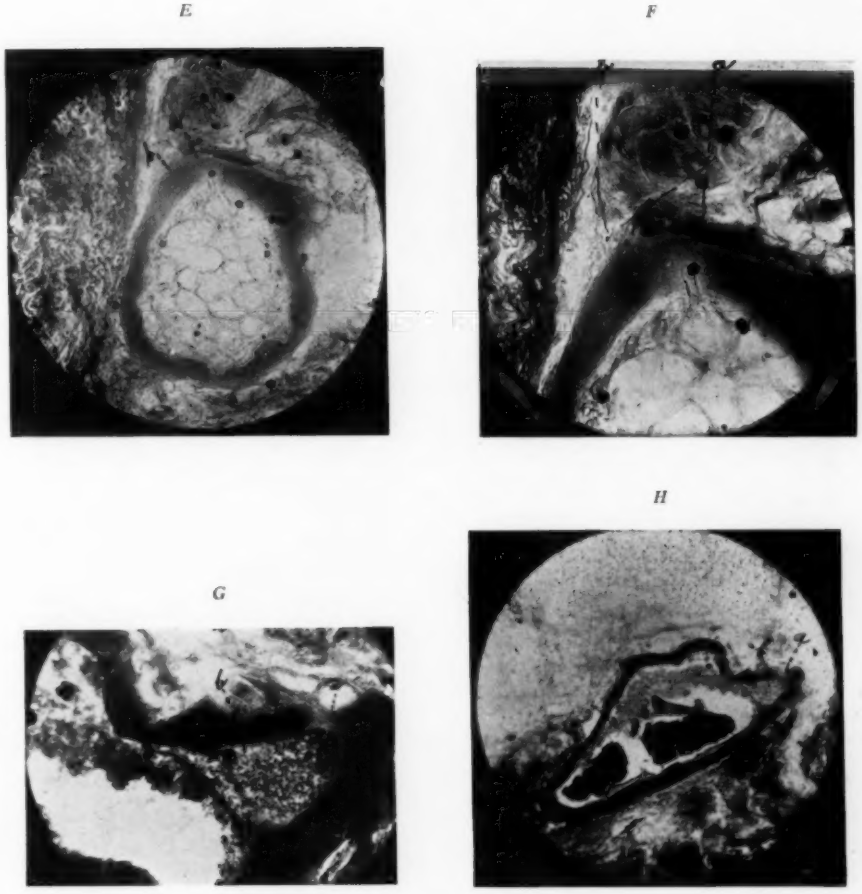


FIG. 36.—Connective-tissue bridge with projection inside. *A*, *a*, connective-tissue bridge; *b*, connective-tissue projection inside ring (oc. 2, obj. 16). *B*, *a*, connective-tissue bridge; *b*, connective-tissue projection inside ring (oc. 2, obj. 4). *C*, *a*, connective-tissue projection toward inside of ring and bridge; *b*, connective-tissue participation in bone formation; *c*, suderiferous gland (oc. 2, obj. 16). *D*, *a*, connective-tissue projection toward inside of ring and bridge; *b*, connective-tissue participation (oc. 2, obj. 4). *E*, *a*, connective-tissue bridge; *b*, connective tissue participation in bone formation outside of ring (oc. 2, obj. 16). *F*, *a*, connective-tissue bridge; *b*, connective-tissue participation in bone formation outside of ring (oc. 2, obj. 4). *G*, a, connective-tissue bridge; *b*, erythrocyte participation (oc. 2, obj. 16). *H*, *a*, connective-tissue bridge, *b*, erythrocyte participation (oc. 2, obj. 4).

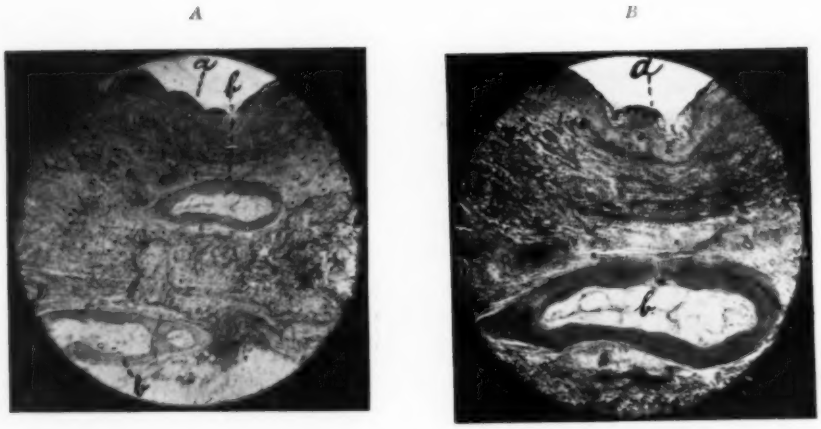
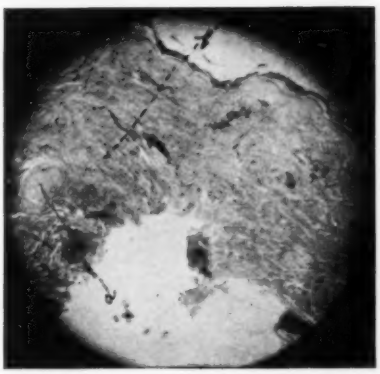
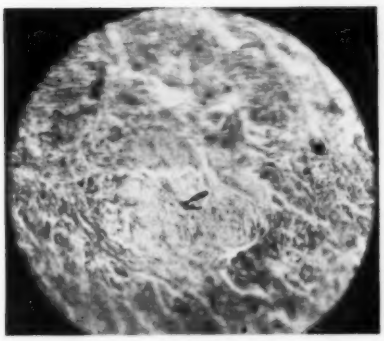


FIG. 37.—Bone rings with empty centre near epidermis. *A*, *a*, epidermis; *b*, emptied bone ring (oc. 2, obj. 2 *). *B*, *a*, epidermis; *b*, emptied bone ring (oc. 2, obj. 8).

A



B



C

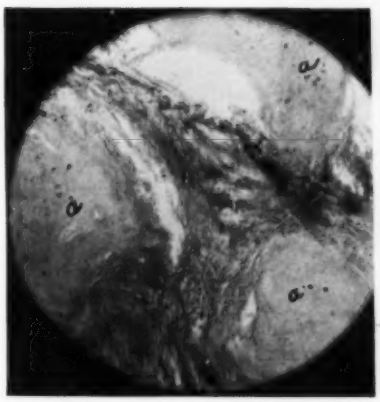


FIG. 38.—Initial hemorrhage at the side of three solidified bone formations, all close to epidermis. *A*, *a*, two of the bone formations; *b*, hemorrhage (oc. 2, obj. 2 *). *B*, *b*, hemorrhage (oc. 6, obj. 16). *C*, *a*, the three bone formations in part (oc. 6, obj. 16).

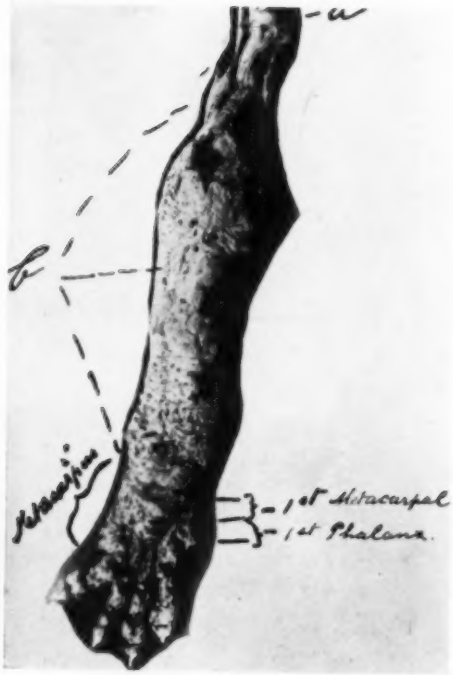


FIG. 39.—Right thoracic extremity of dog (anterior view). *a*, head of humerus in glenoid fossa; *b*, ossifications—compare diminutive first metacarpal and first phalanx of thumb with those of other digits.

FIG. 40.—Right thoracic extremity of dog (posterior view). *a*, head of humerus in glenoid fussa *b*, mass of ossification; *c*, location of thumb anteriorly.

FIG. 41.—Longitudinal section through right wrist and carpal bones.

in his turn was the ancestor of all the other short-fingered progeny, so that in this family the hereditary defect was limited to the side-issue of the hired man.

Philip W. Mathews[94] had the extremely rare opportunity to observe a hereditary true brachydactylia through five generations. All the afflicted members had shortening of the third, fourth and fifth fingers on both hands, with broadening of the third phalanx of both fourth fingers.

Mario Bertolotti[95] gives another very remarkable report of three generations of a brachydactylic family, with ten out of a total of fifteen members showing a brachydactylia of the third fingers; he enters into the developmental history of the metacarpal bones and refers to Meckel, who came to the conclusion that the first metacarpal, judging by its location and peculiar service, and especially owing to its mode of ossification (being the only one of the metacarpal bones which has its epiphysial nucleus like all the phalanges on the proximal side), should be considered the first phalanx of the thumb. Radiography shows that at times one encounters in boys from 5 to 15 years an accessory nucleus on the distal side besides the old classical proximal.

Ludwig Freund[96] published the first of such cases and showed that in about 24 to 30 per cent. an additional distal special epiphysiary nucleus to the usual proximal one in the first metacarpal (also the first metatarsal in myositis ossificans progressiva) may be seen. He also found an occasional extra proximal nucleus in the second and fifth (in our case in the fourth) and that the atrophic character of such nucleus formation shows a regressive anatomical variation.

According to Dubreuil-Chambardel[97] this ossifyng nucleus in a distal epiphysis of the first metacarpal was present in eighty-two cases out of a total of 240 X-ray examinations. It commenced to appear in the third year and the complete ossification was reached at about the thirteenth or fourteenth year.

Freund[98] has observed the accessory variation now and then on the proximal side of the second, and, even more rarely, of the fifth metacarpal bone. These men all believe that the choice of epiphysial nuclei of the metacarpal bones depends upon their separate physiological functions. Therefore the proximal site in the first metacarpal depends upon the adaptation of this metapodical process to the functions of the thumb. To support this hypothesis Bailleul[99] as well as Dubreuil-Chambardel have each published a radiograph of a case of a triphalangeal thumb, where the first metacarpal showed its nucleus of ossification only on the distal side.

According to Koehler[100], however, this anomaly represents a certain regressive type met with in the cetaceæ and other water-mammals, where the thumb and big toe have two epiphysial ossifying nuclei. He also quotes Henle as having demonstrated from comparative anatomy that the metacarpal bones had originally two nuclear epiphyses at the same stage of development, a proximal and distal one, although with the human the rule is the distal one for the four last and the proximal one for the first metacarpal. Cruveilhier and Gegenbaur hold the same opinion. They draw attention to the curious anatomical conformation of certain

[94] A Case of Hereditary Brachydactylia. Brit. Med. Journ., Oct. 31, 1908, p. 969.

[95] Contributi radiolog. allo studio della sciluppo osteogenetico dei metacarpei. La Radiologia medica. Marzo, 1915, vol. ii, Fasc. 3, p. 105.

[96] Ueber Pseudoepiphysen. Ztschrft. f. Morphologie & Anthropologie, vol. viii, 1905, p. 87.

[97] Sur le development du premier rayon digital; Gazette medicale du Centre, p. 288, 1913.

[98] Op. cit.

[99] Ossification du 1r metacarpein. Societe de Biologie. March, 1911.

[100] Vollzaehlige proximale metakarpale Epiphysen. Fortschr. a. d. Geb. der Roentgenst, vol. xix, p. 279, 1912-13.

cetaceæ, balaenidæ and sureniæ, who present double epiphyses of the four last metacarpal and an evident trace of an additional distal epiphysis in the first.

Freund[101] confirmed these statements by his own investigations of various cetaceæ. A double epipyhsial nucleus in human metacarpals appears, therefore, in the light of a reversive type or mongolism.

Koehler[102], who advocates this view, supports it with the report of a unique case in medical literature. A patient 12 years old, suffering from infantile myxœdema, showed, in the X-ray picture of his hand, a development of his carpal bones corresponding to that of a child of three to four years, and all the epiphysial nuclei of his five metacarpal bones were on the proximal side.

Bertolotti supplements this with a radiographic discovery in the course of examination of his above-mentioned family. He found the brachydactylic members showing pseudo-epiphysial basal nuclei of their second metacarpal bones.

We cannot close this discussion of functional adaptation of nuclei of ossification with regard to the first metacarpal bone without adding here an observation pointing apparently to the opposite direction. W. Rieder[103] published the case of a family of eight persons in its third generation, showing bilateral hyperphalangia of thumbs (first metacarpal and three phalanges). They were father, 45 years old; daughter, 19 years old (first marriage), and her two daughters, 12 and 11 years; also her son, 4 years old, all three from her second marriage.

This array of facts refutes, we believe, conclusively any attempt to explain the presence of brachydactylia from exogenic causes, either by amniotic bands or intra-uterine pressure. The overwhelming evidence of the hitherto unused material of myositis ossificans progressiva, showing in practically every true case the accompanying feature of symmetrical brachydactylia of fingers, toes or both, does not seem necessary to prove that defects in the primary " anlage " must be held responsible for this deformity; a view which Burgerhout[104] expresses very strongly in his dissertation.

We cannot enter here into the more subtle questions of mongolism or atavism, which present many tempting reasons for discussion, but stand firmly for the adoption of primary endogenic developmental causes for brachydactylia. To this extent we may expect now to clear the way for a possible later answer to the ever-open question for final causes. Even to the location for the embryonic layer which had been charged with the formative fault—our case furnishes a new aspect. Rolleston[105] as well as Morley, Fletcher[106] and others are united in singling out the mesoblast as the culprit, Rolleston calling it the skeletal layer with its subdivision of the muscular. My case shows bone-formation also in the skin and draws for the first time this outer layer into participation of the pathological process. As our present

[101] Op. cit.

[102] Op. cit.

[103] Eine Familie mit dreigliedrigen Daumen. Zeitschr. f. Morphologie und Anthropologie. 1900, p. 177.

[104] Op. cit.

[105] Op. cit.

[106] Op. cit.

views do not sanction the strict division of embryological layers as the exclusive formative agents of distinct types of tissue, it may perhaps be surmised that the pathogenetic cause exerts its influence at a very early period of embryonic life, before any distinct differentiation has begun.

We believe a similar origin may be claimed for the osteogenetic hemorrhages. In my series of microscopical pictures a gradual progression is shown from the primary hemorrhage into the corium to the osseous growth by either the fibrocellular or hyaline cartilaginous or the osteoblastic route to the final development into bone tissue. So clear is this progress that in some places the pictures appear to indicate a direct metamorphosis of the erythrocytes into the fusiform osteogenetic cells (fibroblasts). The entire course of events from primary hemorrhage to finished bone lies illustrated before our eyes, but we want to know the reason for these small hemorrhages and here we have to recur to the endogenetic fault in the "anlage" and have it extended to the capillary and minor peripheral blood-vessels.

The same developmental disturbances may, on account of the identity or close proximity of the basic tissue for both, easily affect the vascular together with the skeletal systems. The fingers or toes present, as endings of the skeletal system, anatomically similar relations, as the smallest blood-vessels and capillaries, as the last endings of the vascular system, in the integumental parts. A defective wall-construction of these two may account for a considerably lessened resistance in the integumental tissue, skin and muscular, to slightest traumatism, or variation of blood-pressure, and a consequential easy rupture with resultant circumscribed hemorrhages. Thus an acceptance of the theory of a simultaneous "anlage-defect" in the vascular as well as in the skeletal system may give us a better understanding for the causes underlying the early stages of the bone-forming process in this disease, which our microscopic sections so fully illustrate. We shall not go any farther in our explanatory attempts and refuse to go "a'sailing upon unknown seas" with Maunz[107], who, endeavoring to explain his views of formation of bone, asserts that with the anlage of the muscular system and the osseous skeleton, embryonic osteoblastic tissue-germs have been wandering about and deposited in such places of the muscular connective tissue, tendons and fasciæ, where later the osseous formations start. Nor shall we enter into a discussion with the other theorists, such as Goeschen[108], who, after referring to the peculiar calcium metabolism (?) of the myositis ossificans patients as stimulating the ossifying process, finally adds: "Up to date the acceptance of a disposition to such ossification cannot be entirely dispensed with." While we have offered a tentative explanation of the pathological anatomical development in line with the microscopical pictures, we must admit our complete ignorance of its more remote causes.

The therapeutic part of our theme is very brief. In the early period

[107] Op. cit.

[108] Ueber Histologie & Pathogenese der circumscripten Muskelverknoechrg. (Jena 1913. Gustav Fischer.)

of its history, the disease was attacked with all sorts of remedies, alteratives, solvents for calcium supposedly retained in the system by faulty metabolism, the inevitable iodide of potassium, etc.; every one of them with more or less marked success observed solely by its original author but pronounced a complete failure by every other follower. The favorable result of thiosinamine injections reported by Boseck[109] has never been achieved by anyone else; moreover, his alleged case of myositis ossificans progressiva lacks every convincing feature of deserving such classification. In myositis ossificans traumatica Aizner[110], Nicolai[111] and Grosskurth[112] have reported favorable action of the same drug (fibrolysin). This disease, however, disappeared often spontaneously, so the therapeutic effect should not be unreservedly endorsed. Lately Nové-Josserand and René Horand[113] have earnestly recommended an X-ray treatment for the progressive form. They report excellent results in their case by a series of twenty-six treatments, all below five Holzknecht. I had the abdomen of my case treated in this way without the least effect, except perhaps to stimulate new growths.

In summing up, the conclusions of this treatise are as follows:

CONCLUSIONS

1. Myositis ossificans progressiva is a misnomer for a pathological process of atopic ossification starting primarily in the connective tissue of the exoskeleton and involves in its further progress the tissues of the endoskeleton. It would better be named fibrocellulitis ossificans progressiva. The disease nearly always commences at an early period of life and shows as additional characteristic symptoms a microdactylia of fingers and toes, and in some cases a more or less pronounced degree of infantilism.

2. Similar to the primary hemorrhage of the traumatic myositis ossificans, the introductory feature of the pathological process in the progressive form is an initial intercellular capillary hemorrhage which starts the growth of newly-formed connective tissue surrounding the hemorrhagic extravasation without any previous hypertrophy of the adjacent connective tissue.

3. Calcification and ossification take place at first at the newly-formed connective-tissue strands encircling the hemorrhagic mass, and with few exceptions from within outward.

4. There is an apparent active participation of the enclosed erythrocytes in the bone-formation; they change their round form to a spindle-cell shape and seem to become nucleated as they approach the osteogenetic edge of the encircling ring and merge into its substance.

5. The microdactylia has an endogenetic origin and is the result of an "anlage" defect of the carpal and tarsal tissue rays ("gewebsstralen"),

[109] Zur Heilg. der Myositis ossificans progressiva geheilt durch Fibrolysin. Muench. mediz. Wochschr., 1906. p. 2350.

[110] Muench. mediz. Wochschr., vol. lvi, part 1, p. 756.

[111] Deutsche militaeraerztl. Zeitschr., Heft 18, 1907.

[112] Deutsche militaeraerztl. Zeitschr., Heft 18, 1908.

[113] Op. cit.

causing thereby an imperfect development of the last endings of the extremities in one part or other.

6. The initial hemorrhage is probably due to a similar cause affecting the integrity of the last endings of the vascular system, the capillaries, from where the hemorrhage starts.

7. The calcification and consequent ossification of the newly-formed connective tissue is due directly and indirectly to the primary hemorrhage. The hemorrhagic effusion with its calcium content starts the first deposit of lime and indirectly stimulates its continuation by a nutritive impairment of the surrounding tissue from pressure of the extravasation.

8. Investigations made under scientifically correct premises show there is no reason for believing in a diminished calcium excretion or a calcium retention in this disease.

9. No therapeutic measures against the products or the progress of the disease have proven of any value.

An agreeable duty has yet to be discharged. My heartiest thanks are due Miss Ophuls and Mrs. Hurwitz, the librarians of the Lane and U. C. Libraries, for their untiring help in providing the literature from their own and the Surgeon General's library; to Doctor Israel, from the latter institution, for the translation of the Slavic papers; and to Mr. Ginsburg, the roentgenologist of the Mt. Zion Hospital, for his patient and indefatigable photographic work.

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THE DANGERS OF UTERINE CURETTAGE*

By W. A. Lincoln, M.D., F.A.C.S. (Eng.)
of Calgary, Alberta

The curet, in the shape of a scoop, was first invented and used by Récamier in about 1845. The sharp curet was introduced by J. Marion Sims in 1865, and the dull curet by T. Gaillard Thomas in 1880. Since its introduction it has been very universally accepted in the surgeon's armamentarium and used with the greatest freedom, the most accepted rule of conduct being "when in doubt curette," more especially as the pathologist offered moral support in the shape of a beautiful classification of the uterine scrapings into glandular and interstitial endometritis, one or other of these conditions being practically always found, or in case it was not very distinctive, it was called a "mixed condition," both elements being present. A nice symptomatology was built up around these all the way from amenorrhœa, dysmenorrhœa, metrorrhagia, menorrhagia, or any other kind of a discharge, to general symptoms of lassitude, headache, backache or any other ache almost anywhere. In case some women might be missed out, it was stated that it might be present without any symptoms whatever. Given a curet and a woman, the old-time gynæcologist would always find endometritis. In 1908 Alder and Hitschman[1] demonstrated that what has been classified as endometritis was really nothing more than stages in the usual menstrual cycle, the premenstrual stage corresponding to the old glandular endometritis, and the postmenstrual stage to the old interstitial endometritis, neither of which was pathological. Cullen, in the examination of a great many specimens, has demonstrated that endometritis is really a very rare disease. It has also been shown that where endometritis really exists hemorrhage is not a symptom.[2] Furthermore, uterine hemorrhage is rarely cured by a curettage. Busse collected five hundred cases so treated, and only found benefit in 10 per cent. Thus we have had to forget all we were taught about endometritis and start over again. But old ideas die hard, and even in some fairly modern textbooks you will still find a good deal about endometritis, while the average practitioner clings to his curet with the fondness of an old friend. Were it only ineffectual as a remedial agent not much serious harm would result, but that it is fraught with grave dangers has been long recognized. In puerperal infections many deaths have been laid to its door. The dangers in this condition have been so emphasized that happily it has now been pretty well abandoned, but it would seem that its dangers in other conditions have not been sufficiently emphasized. A. P. Heineck[3] collected and analyzed 160 cases of perforation of the uterus occurring during the course of intra-uterine instrumentation, while Schweetzer[4] from 1911 to

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1915 was able to find 105 cases reported in the German literature, with a mortality of 25 per cent. I am sure that these figures do not give an accurate estimate of the extent of accidents following the use of this instrument. The successes find their way into the medical literature, the failures are silently buried—sometimes underground. In addition to dangers of rupture, dangers of sepsis and hemorrhage are of considerable importance. In order to get some idea of the difficulties being encountered I sent a circular letter to some of the men practising in this province, and from 24 men received the following 43 unreported cases with 11 deaths—a mortality of about 25 per cent. The names of some of the doctors are omitted by request.

I. *Cases of Hemorrhage.*—This I believe is a fairly common complication. I am reporting five of the more severe cases with three deaths, although I have other case reports, and I am sure nearly everyone has seen cases of severe hemorrhage, which ultimately recovered.

Case I.—Multipara, aged twenty-eight years, confined two weeks previously by a midwife. Had some brownish discharge, a temperature of 101, and a pulse of 120. Some pain and tenderness over the lower abdomen. Curetted and some pieces of placenta brought away. Hemorrhage became very profuse and uncontrollable, and the patient died soon after being returned to her bed.

Case II.—Multipara, aged twenty-eight, two and one-half months pregnant. Incomplete abortion, for which a curettage was performed. Particles of placenta removed and considerable hemorrhage encountered, but seemed to be controlled. Surgeon and anæsthetist had left the patient, when she again began to flow, and died before help could be obtained.

Case III.—Primipara, aged twenty-three, pernicious vomiting, for which the patient was curetted. Much hemorrhage was encountered and the patient died six hours later. It is possible that acidosis had some influence in this case, but the attending surgeon is of the opinion that the hemorrhage was the cause of death.

Case IV.—Reported by Dr. E. Hobart Reed, Calgary. Multipara, aged thirty-five years, very severe hemorrhage following each confinement. Incomplete abortion at two and a half months. Os found dilated, and in attempting to remove placenta very severe hemorrhage was encountered. Uterus had to be packed to save patient's life. Eventually she recovered, and at a later date had a resection of the tubes.

Case V.—Reported by Dr. E. A. Garner, Fernie, B. C. Miscarriage at four months and was having considerable hemorrhage, curetted by the attending physician, who encountered severe hemorrhage and packed the uterus. Five days later she had another very severe hemorrhage, and was curetted without an anæsthetic. Condition very low, but she eventually recovered.

II. *Cases of Sepsis.*—This may be introduced, but more commonly is present, perhaps in a mild form, and is lighted up by the curettage. A curettage in puerperal infections is particularly fatal, and I believe at present

very rarely done, although I quote one case. I have other reports of mild infections following this operation, but only report eight of the most severe, with three deaths.

CASE VI.—Primipara, aged thirty years, difficult labor. Rise of temperature on the fourth day with some odor to the discharge. Patient anæsthetized and curetted, following which she had a chill and very severe septic symptoms from which she died about ten days later.

CASE VII.—Reported by Dr. E. S. Garner, Fernie, B. C. Mrs. L., three months pregnant, miscarriage two days previously. Temperature 100, pulse 130. Curetted by the attending physician, with the removal of a small amount of placental tissue. Patient ran a septic temperature for about a week, when an abscess was evacuated from the pouch of Douglas. Patient eventually recovered.

CASE VIII.—Multipara, aged thirty-two years, two and one-half months pregnant. Pain and irregular hemorrhage for a week, after which she was curetted; three days later she took a severe pain with a chill and a rise of temperature to 104 and pulse to 130. She continued to run a septic temperature for some days, when about a pint of pus was evacuated from the pouch of Douglas, after which she made a good recovery.

CASE IX.—Multipara, aged twenty-seven years, normal confinement with a somewhat slow recovery, after getting about continued to have a bloody discharge. Curettage done by the attending physician, at which a moderate amount of blood was lost, and shortly afterwards she had a very severe hemorrhage, became blanched, pulseless, and nearly died. A few hours later she had a severe chill, and temperature rose to 104. She continued very ill with a severe septicæmia for several months, but eventually recovered.

CASE X.—Primipara, aged thirty years; three months pregnant, symptoms of an incomplete abortion, with a temperature of 102; curetted and particles of placenta removed. Developed a pelvic cellulitis and ran a temperature for several weeks. Later she developed a subphrenic abscess, which was opened, and the patient recovered after being ill for four months.

CASE XI.—Multipara, aged thirty years; miscarried at three months. Twice curetted by the family doctor. Ran a high temperature and looked very ill. Uterus explored and particles of placenta found to be still present. Streptococci found in the blood. Patient died about a week later.

CASE XII.—Multipara, aged thirty-seven years; badly lacerated cervix, two months pregnant; self-induced abortion. Chill and a temperature of 104; under rest this subsided, but returned later, and the patient was curetted by another doctor. She then developed a typical septicæmia and died in about a week's time.

CASE XIII.—Primipara, aged twenty-four years; normal labor. Temperature rose to 104 on the fourth day; light curettage performed, after which she ran a normal temperature for two weeks; temperature then rose to 102, together with severe abdominal pains. The abdomen

was opened and an abscess found in the outer wall of fundus of the uterus, which had ruptured into the peritoneal cavity. This was drained and the patient made a good recovery.

III. *Cases of Rupture.*—I have collected twenty-seven of these cases, with four deaths. Rupture may be of the cervix, the body or the fundus, and may be made by dilators, sound, or curet.

Case XIV.—Reported by Dr. J. H. Gillespie, Picher Creek, Alta. Primipara, aged twenty-six years, with a badly ruptured cervix, where a curettage had been previously performed for sterility. The operation did not produce the desired effect, but instead gave the patient a lot of new trouble. I have other cases where the cervix has been badly lacerated, but the results are usually not serious, and I merely quote this one as an example.

Case XV.—Multipara, aged thirty-five years; miscarriage, probably induced, some sepsis present. Curetted with a blunt curet. Uterus perforated and a piece of bowel brought out. This was pushed back and the cavity of the uterus packed with iodoform gauze. Patient recovered in about two weeks.

Case XVI.—Multipara, aged thirty-five years; miscarriage. Some signs of sepsis present. Curetted with a dull curet. Uterus perforated and a piece of bowel brought out, replaced, and the cavity of the uterus packed with iodoform gauze. Recovered in about four weeks.

Case XVII.—Reported by Dr. C. W. Holmes, Edmonton. Multipara, aged twenty-six years. History of irregular bleeding for three months, miscarriage taking place at the fourth month. Large amount of placenta removed with the curet, when with no undue force the uterus was perforated. There was no sign of undue hemorrhage and as conditions were unfavorable for abdominal section, patient was left and no ill result followed.

Case XVIII.—Reported by Dr. T. M. Campbell, Lethbridge. Multipara, aged forty-four years. Not a very robust woman, but not suffering from any organic disease. Two months pregnant. Had had a dark bloody discharge for a month. Temperature 99.8. Uterus large, soft and flabby, with a patulous os from which issued a dark foul discharge. Patient was curetted, every aseptic precaution being taken, but inside of a week the patient died of septic peritonitis.

Case XIX.—Reported by Dr. J. E. Lovering, Lethbridge, Alta. Multipara, aged thirty-three years; two months pregnant. Stated that another woman had passed an instrument on her. She was flowing profusely, but no temperature. A curettage was done and a large amount of débris removed. With no undue force the curet suddenly passed into the abdomen. The abdomen was immediately opened and a rupture found on the right side of the fundus, which was repaired. Appendix and a right ovarian cyst removed. Gall-stones were found and removed by a separate incision. Patient was in a critical condition for several days, but then made a good recovery. Dr. Lovering is of the opinion that the rupture must have been made during the attempted abortion, as he used very little force in his manipulations.

CASE XX.—Reported by Dr. W. S. Galbraith, Lethbridge. Case that was prepared for an abdominal operation, a curettage being done as a preliminary procedure. At the subsequent operation the rupture was repaired with no ill effect.

CASE XXI.—Reported by Dr. W. S. Galbraith, Lethbridge. Where he operated upon a case 100 miles from home, which proved to be an ectopic, and in which the local doctor had curetted and perforated the uterus. There was also a severe infection present, and the patient subsequently died.

CASES XXII and XXIII.—Dr. F. W. Gershaw, Medicine Hat, reports that in two cases he has perforated the uterus with a curet, but in each case he had planned to do an abdominal operation when the rent was repaired, with no serious after effects.

CASE XXIV.—Dr. J. S. McLeod, Medicine Hat, reports that while an interne in an American hospital and doing his first curettage under the direction of the chief surgeon, in a case where the uterus was subinvoluted and friable, a sharp curet was used with considerable care, and suddenly, without any undue force, the uterus was perforated. Abdominal section immediately performed and a rupture found on the anterior wall of the uterus, which was repaired, and the patient made a good recovery.

CASES XXV and XXVI.—Dr. C. E. Smyth, Medicine Hat, reports that he has seen two instances of rupture of the uterus during curettage. In both instances the abdomen was opened and the rupture repaired, with no resulting ill effects.

CASE XXVII.—Multipara. Profuse menstruation and some bleeding between periods. Anæsthetized and the sound passed to explore the interior of the uterus, and this with no undue force passed into the abdominal cavity. No further manipulation was carried out, and the patient made a good recovery.

CASE XXVIII.—Multipara, two and one-half months pregnant. Flowing for several days. Anæsthetized and os dilated with heavy dilators. Placenta forceps inserted and a piece of bowel brought out. This was pushed back and the abdomen immediately opened and a large rent found in the anterior wall of the uterus. This was repaired and the patient made a good recovery.

CASE XXIX.—Curettage on a multipara for irregular hemorrhage. Large dilator used, and this seemed to enter in a forward direction. On investigation it was found that the front wall of the uterus had been ruptured. Patient was put to bed, but had extreme pain, vomiting and a rising pulse-rate. Abdomen was then opened and a rent in the anterior wall of the uterus repaired. Drainage inserted and the patient made a good recovery.

CASE XXX.—Multipara, a German woman with a large, flabby uterus, perforated by a curet with very slight force. Abdomen immediately opened and the rupture repaired. Patient made a good recovery.

CASE XXXI.—This case was witnessed by the reporter in a foreign clinic, where a professor with an international reputation was demonstrating a special dilator, which goes by his name, when the cervix and

anterior wall of the uterus was badly ruptured, resulting in a profuse hemorrhage and death of the patient.

CASE XXXII.—Reported by Dr. J. S. Wright, Edmonton. Multipara, aged thirty-two years. History of five miscarriages. Endometritis with purulent discharge. Dilated and curetted with a moderately sharp curet and with no undue force. Curet seemed to pass to an unusual depth. Irrigation instituted and the fluid did not return. Again explored and rupture located. Abdomen immediately opened and rupture repaired. Patient made a good recovery.

CASE XXXIII.—Aged thirty-five years, no living children, but repeated miscarriages. Was three months pregnant. Complained of a purulent, blood-stained discharge present for a week. Dilated with difficulty, and a macerated mass removed with the dressing forceps. Uterus foul and soft, so that curet passed through without knowledge. Patient died four hours later, and the post-mortem examination showed a softened uterine wall with a rupture on the right side involving the uterine artery. Syphilitic nodes were found throughout the body.

CASE XXXIV.—Reported by Dr. J. W. Auld, Calgary. Multipara, aged thirty-four years. Abortion at three months a short time previously. Double salpingitis, for which the patient was prepared for operation; preliminary curettage with a sharp curet. Without undue force fundus of the uterus was perforated. At the abdominal operation the tubes and uterus were removed, and the patient made a good recovery.

CASE XXXV.—Primipara, dilated and curetted for dysmenorrhœa. Sound passed through the anterior wall of the cervix instead of through the internal os; this was followed by dilators, and it was then found that rupture had taken place. Packed with iodoform gauze, and the patient made a good recovery.

CASE XXXVI.—Multipara, aged thirty years. Not pregnant. Irregular bloody discharge, and the patient thought she was pregnant, and passed an instrument upon herself. A few days later patient was anæsthetized and sound passed without any obstruction. Large dilator inserted into the same passage and dilated. Forceps inserted and a piece of bowel brought out. Abdomen immediately opened and a rupture found in the posterior wall of the uterus. This was repaired and the patient made a good recovery. Surgeon in charge was of the opinion that the rupture must have been made by the woman.

CASE XXXVII.—Multipara, aged thirty-seven years; two months pregnant; had been flowing for a month. Uncertain whether the fœtus and placenta had been expelled. Anæsthetized, and the sound passed. This without any undue force perforated the uterus. Packing inserted, and patient made a good recovery.

CASE XXXVIII.—Multipara, aged thirty-three years. Two and one-half months pregnant. Admitted having passed an instrument some time previously, and was flowing freely. Dilated, and pieces of placenta removed. When finally exploring with a dressing forceps a piece of bowel was brought out. This was returned and the uterus packed. The patient made a good recovery. The surgeon in charge is strongly

of the opinion that the rupture must have been made by the woman herself.

CASE XXXIX.—Multipara, aged thirty-eight years. History of an abscess with purulent discharge after the second child. Miscarried at the second month. Os dilated with graduated dilators, and the direction of these did not seem to be correct. An investigation showed that the posterior wall of the cervix had been ruptured and the instruments were passing up into the abdominal cavity. Packing was inserted and the patient made a good recovery.

CASE XL.—That late results may follow rupture is shown by the report of a case by Dr. E. W. Allin, Edmonton, where intestinal obstruction followed an old rupture of the uterus.

Many men reported that they were positive the rupture must have been previously produced, as they used so little force, yet I saw some of these cases and they did not suffer from any vomiting or abdominal pain previous to the operation, and the rupture when repaired was recent. I am of the opinion that it should rather indicate how easy it is to produce a rupture. The treatment of this condition when produced should depend upon the circumstances. If the case is clean and the rupture a small one it is better to leave it alone and no ill results will follow. If, on the other hand, the rupture is large, or if there be sepsis, the abdomen should be immediately opened and the condition rectified.

IV. *Cases of Curettage in Pernicious Vomiting.*—Here the result may be largely due to the underlying disease, but the curet may be an added factor, and is extremely dangerous, as the following cases demonstrate.

CASE XLI.—Reported by Dr. C. U. Holmes, Edmonton. Primipara, aged twenty-four years. Six weeks pregnant. Developed vomiting of a pernicious type and lost 40 pounds under treatment. After consultation patient was curetted. She came out of the anæsthetic, but the pulse remained weak, and she died 12 hours later.

CASE XLII.—Also reported by Dr. Holmes. Primipara, once previously curetted for pernicious vomiting. Again curetted for the same condition. Collapsed at the beginning of the anæsthetic, but picked up and the operation completed. Patient recovered.

These cases are undoubtedly due to acidosis, but the added burden of an anæsthetic and the use of the curet is often sufficient to endanger their lives.

A curettage may be carried out when it is indicated, and yet fail to produce the desired result, as the following case will show:

CASE XLIII.—Primipara. Two and one-half months pregnant, threatened abortion, and it was not known whether or not the fœtus had come away. Curetted and placenta brought away. Irrigated and the patient returned to bed. The next day a fœtus was passed which had escaped the curet. This could not have occurred if the finger or placenta forceps had been properly used.

Thus it is plain that quite a formidable list of accidents with a high death-rate is easily compiled. While some of the bad results may have been attributed to the underlying disease, the curet has to bear the principal blame. My object in reporting these cases is to again emphasize that any intra-uterine instrumentation is a dangerous procedure. The indications for such work practically narrow down to the removal of some products of conception or of bits of tissue for microscopic examination, and even here it is probably better to use the finger or placental forceps in the first instance, and it is somewhat doubtful whether the latter procedure is justifiable unless prepared for immediate removal. A curettage is a major operation not to be undertaken except under the very best conditions, and with every possible precaution, by a careful and skilful surgeon.

My thanks are due to the colleagues who so kindly placed their notes at my disposal.

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SURGICAL TECHNIC IN ORTHOPÆDIC SURGERY*

By Walter G. Elmer, M.D.,

of Philadelphia

Instructor in Orthopædic Surgery in the University of Pennsylvania

Orthopædic surgery makes peculiar demands upon us in regard to surgical technic. The operations, if they are to be successful, must be carried out in an aseptic manner. Perfect asepsis cannot be attained, but we can very nearly reach it if we go about our work with intelligence and painstaking care in regard to the smallest detail.

In performing the operations of orthopædic surgery it is frequently necessary to use a good deal of force in correcting the deformity, and no matter how carefully the skin has been prepared, these forcible manipulations will force the staphylococcus epidermis albus from the deeper layers of the epithelium out to the surface where the organism is capable of causing mild infection in the operation wound. An example of this is seen in the correction of a talipes equinovarus, where the foot is stretched and manipulated by the operator's hands or molded over the König block or perhaps the Thomas wrench is used. And in order to secure the required amount of correction, it may be necessary to divide the posterior tibial tendon by an open incision and do a plastic operation on the Achilles tendon. An operation of this character is usually performed at one sitting. In another class of cases the forcible stretching and correction is done at some time previous to the operation and the foot held in its corrected position in a plaster-of-Paris case until a later time, when the tendon transplantation or arthrodesis may be done—as in the infantile paralysis cases.

When silk is imbedded in the tissues and we wish it to remain permanently, the slightest infection of the silk will defeat the operation, as the silk will eventually cause suppuration and must be removed. If silk is used as an artificial ligament or tendon, it acts as a foreign body in the tissues. The tissues react to its presence and deposit granulation tissue along the strand. This in time becomes converted into fibrous tissue and we have then a new ligament or tendon of living tissue, the centre of which is the buried silk. The process requires a long time. The limb must remain fixed in plaster of Paris about nine months. The silk will then remain permanently in place. But if the silk is infected with even so mild an organism as the staphylococcus of the skin, it will probably loosen and work its way out or must subsequently be removed.

In opening joints great care must be taken to avoid introducing the

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skin coccus within the joint cavity. Therefore, two knives should be used—one for the skin incision and another to use in the deeper structures.

But my purpose in presenting this paper is to call attention to several important ways in which the wound may become contaminated by organisms far more potent than the skin coccus, types of infection which may defeat the operation and even be a serious menace to the life of the patient. The responsibility rests largely upon the nursing staff and the technic of the operating room is good or bad according to the intelligence and ability of the nurses in charge of it.

The head nurse must directly and personally supervise the work of her assistants, and the directress of nurses is responsible for the head nurse. The most important feature of a large general hospital is the operating room—and this includes, of course, its personnel.

The visiting surgeon in performing a series of operations expects everything to proceed smoothly and without friction—doctors and nurses working in harmony—and all coöperating to secure the best results. When one operation follows another in quick succession—perhaps not five minutes in the interval between them—it is not always possible to relegate the septic cases to the last, as it sometimes happens that infectious material is encountered when it is not expected.

A gall-bladder may be septic or an appendix may be lying in a small pocket of pus. A clean pelvic operation may reveal a pyosalpinx. Therefore, in every series of operations one must take it for granted that they may not all be clean cases.

The operating-room nurse and her assistants must have absolute confidence in their ability to so conduct the technic that there is no possibility of carrying infection from one patient to another. This requires constant vigilance and can only be entrusted to a highly trained head nurse.

In most hospitals it is customary for the directress of nurses to make a daily tour of inspection of the hospital under her charge. She visits the private patients and the ward patients, sees that the wards are clean, looks at the bed linen, walks into the kitchen and pantry, opens the doors of cupboards and closets, inspects the toilet rooms and in a hundred other ways assures herself that the hospital is being conducted in a clean and orderly manner. And yet more important than all these is the operating room, and I would suggest that the directress of nurses occasionally vary her routine and go unannounced into the operating room or the clinical amphitheatre when a series of operations is in progress and remain throughout an entire forenoon or afternoon watching with vigilant and critical eyes every detail of the work of her nurses.

In my visits to hospitals in other cities I usually seek out the operating room nurse and, if she can spare the time, ask her many questions in regard to the surgical technic.

From a seat in the clinical amphitheatre during a series of operations

one can also gain a very fair idea of the care and thoroughness with which the nurses have been taught.

I will mention some of the weak links in the chain of surgical technic as they have come to my notice in different hospitals, and the fact to be kept in mind is that any one of these weak links is capable of causing a complete breakdown in our surgical asepsis and result in the failure of our efforts to secure clean primary healing of our operation wounds. Of what use is it to insist on our surgical staff, both doctors and nurses, wearing mouth guards, when it is possible to point out faults in the technic by which septic virus may be carried from an infected case to a clean one?

First, then, we will consider the gutta-percha gloves. As the gloves can be sterilized *absolutely*, it is a good thing to use them. But the most important function of the rubber gloves is to prevent the skin of our hands from being infected with the highly tenacious poison of a septic case, as the skin of our own hands when so contaminated cannot be rendered clean for a clean operation which is to follow. A doctor or a nurse may carry this infection on their hands for several days in spite of all efforts to disinfect them. And it is most important that the hands and forearms of the surgeon and his staff of assistants should at all times be protected from contact with septic material.

The preparation of the rubber gloves, therefore, is a matter of the most vital moment. Beginning, then, with a pair of gloves which have been worn during a septic operation—which might have been a ruptured appendix and local peritonitis, an empyema of the gall-bladder or of the thorax, a pyosalpinx, or a dermoid cyst, drainage of an infected knee-joint or opening the thigh-bone for acute osteomyelitis, puerperal sepsis and many other conditions. Poison of this character may remain potent for many days upon rubber gloves, basins, table tops and the like.

The infected gloves are washed with soap and water by a nurse who perhaps is handling them with her bare hands. Her hands, therefore, become the carriers of infection, and even though she may not be assisting at operations, she may have a good deal to do with making the necessary preparations for an operation.

In some hospitals the nursing staff apparently has implicit faith in the autoclave. The nurses believe—and it is difficult to convince them to the contrary—that everything that comes out of an autoclave must be sterile because it has been exposed to live steam for twenty minutes or a half hour. But the autoclave is fallible. There is a curious and inexplicable inconsistency about the use of the autoclave. The gauze and cotton which come to the hospital from mills or factories, where it is most unlikely they could have become contaminated by any really virulent organisms—probably nothing more than the ordinary dust of a workroom, which is relatively harmless—are put into the autoclave for a half hour on three successive days in order to destroy all germs and spores. The most harm-

less of all the materials used at the operation are subjected to the most rigid and thorough sterilization.

The live steam under twenty pounds pressure penetrates every portion of the cotton, gauze, bandages, sheets, towels, gowns, etc.

And now as to the rubber glove. It is probably capable of greater harm than any other article which is used at the operation. Operating-room nurses have sometimes told me that they depend upon the autoclave to sterilize the gloves. If the gloves have been used in a septic case they are sterilized for twenty minutes, and if they have been used in a clean case they are sterilized for ten minutes. How the nurse knows whether a case is a clean or a septic one I do not know, because it sometimes happens that the operator himself does not know, and only a laboratory report by the bacteriologist can decide the point. When the nurse is asked why the gloves used in a clean case are sterilized only ten minutes instead of twenty, she replies that the longer exposure to the live steam is harmful to the rubber—that it shortens the life of the glove. She admits that the twenty-minute period is desirable for the septic gloves, but she does not and cannot know whether the gloves are septic or not in some cases. The gutta-percha is impervious to steam. The gloves are sometimes folded twice upon themselves and bound up in a small muslin package and a pile of these are packed into the autoclave. Now it is entirely probable that the live steam reaches all the parts of the outer surface of the glove, but I believe there are air pockets inside the glove—probably in the fingers or thumbs—which the steam never reaches. These air pockets therefore permit only *dry heat* sterilization instead of moist heat sterilization for twenty minutes. And the nurse knows that she is dealing with a glove which has been used in a case which was frankly a septic one. She runs her autoclave at about twenty pounds pressure. This provides a temperature of approximately 260° in the sterilizing chamber. This is moist heat sterilization.

We know that boiling water (210° F.) will destroy all organisms and their spores in five minutes. The nurse therefore believes she has a wide margin of safety. But she overlooks the air pockets inside the gloves. These are receiving only dry heat. In order to destroy all germs and their spores by dry heat an exposure of about one hour at a temperature of 350° F. is required. The autoclave falls short of this by nearly 100° in temperature and forty minutes in time.

The surgeon, on putting on his gloves, may find when he opens the package that he has two rights or two lefts through an oversight on the part of the nurse who prepared them and proceeds to reverse one of them, thus bringing the surface of the glove which may not be sterile outside, in contact with the operation field. Or, during the operation the finger of the glove may be punctured or torn and the result may be the same. In order to avoid all possibility of doubt as to the glove being sterile, we have the nurse wash the gloves with soap and water, turning them inside out while

doing so. They are then filled with water to remove the air and immersed under the surface of boiling water and held down by a piece of wire screen so that they cannot float up to the top and be exposed to the air.

They are boiled five minutes by the clock. When the water cools, the nurse, wearing sterile gloves, removes them, dries them with a sterile towel, powders them inside and out with sterile talcum powder and folds back the gauntlet. Into this she tucks loosely a small gauze pad covered with talcum powder which the surgeon uses for dusting his hands. The gloves are then placed without folding in a muslin cover and put into a large glass jar. The final preparation is just before they are needed for an operation. The muslin packets are placed full length in the autoclave, lying loosely in rows, not packed together in compact bundles, and sterilized for twenty minutes. The steam easily reaches every part of the glove and the dusting powder also. The surgeon can have absolute confidence in these gloves—there is no possibility of their carrying septic material from a previous operation.

There is another object which may be a carrier of a deadly virus and that is the sand pillow. It usually has a rubber or mackintosh cover. When it is used in a septic operation—as in an acute osteomyelitis or the drainage of an infected joint or necrosis of bone—the discharges from the wound soak through the sheets or towels and soil the sand pillow. The stains are wiped off with a wet cloth later before the nurse puts it away upon a shelf, but no attempt is made to sterilize it. Within a day or two the pillow may be called for again. This time the surgeon is going to remove a bone graft from the tibia to be inserted into the spine, or he finds it a convenient support in doing an arthrodesis on the foot. The most rigid asepsis is required. A nurse brings in the sand pillow, the surgical nurse wraps a sterile cloth about it and it is placed under the patient's limb. So long as the sterile cloth remains dry no harm results. But it does not remain dry. Blood may run down upon it from the wound, or wet gauze sponges come in contact with it, instruments which have been rinsed in the basin of sterile water may be placed upon it, the surgeon washes his gloved hands in the sterile water and returns to the operation with his gloves dripping, and so the coverings of the sand pillow become wet. It is then only a matter of five or ten seconds before the operation field becomes contaminated with the poison of the septic case of the day before. I usually demonstrate this to my class of students by making a red ink stain on the sand pillow and allowing it to dry. The pillow is then covered with a white cloth and a wet gauze sponge is dropped upon it. In from five to ten seconds the red stain is seen coming through and by the end of two minutes the surrounding areas are red and the gauze sponge stained through and through, although it is fourteen layers of gauze in thickness. The demonstration is very simple and absolutely convincing. This same principle applies, of course, to the tops of the tables upon which the instruments are placed and also the top of the operating table. Very often this latter is covered with a rubber

pad and this in turn with a clean sheet. If the operation happens to be upon a patient's lower limb, the limb is painted with a 3 per cent. solution of iodine while an assistant holds it up with a sterile towel. Then the surgical nurse covers the operating table with a sterile cloth—probably folded to make it double thickness and the limb is put down upon it. If the rubber pad has been soiled from the discharges of a septic case, our clean operation will almost certainly be infected as soon as the table coverings get wet. When one considers the character of the operative cases which come and go in the general routine of the operating room of a large general hospital, the great care which must be exercised by the nursing staff must be apparent.

In a single week there may be a series of operations which includes an operation for gall-stone complicated by an acute septic cholecystitis, the removal of a pyosalpinx, removal of a papillomatous ovarian cyst or a dermoid cyst, a child with ruptured appendix and acute peritonitis, a child with mastoid abscess, another with empyema, and many other similar cases, and all along the clean cases are being operated upon. It is an advantage to have one operating room set apart for septic cases, but even this does not overcome the difficulty. However, the measures to avoid carrying infection from one case to another are simple. There should be a rubber cover provided for each table. They should be sterilized just as the gloves are. The rubber cover is in turn covered with a sterile cloth. The same is true for the sand pillow. The operating table may be covered with a sterile folded blanket and on top of this the sterile sheet folded double. Or a sterile rubber cover may be placed over that part of the operating table which is in the neighborhood of the operation and upon this the sterile folded sheet.

This same procedure is followed for each operation.

The instruments are sterilized for ten minutes by boiling them in water to which a tablespoonful of carbonate of soda has been added. Only the instruments which will be required for the operation should be prepared. It is a disadvantage to sterilize a large number of instruments which are not likely to be used. They unnecessarily complicate the use of the instrument table, and it is also hard on the instruments. The knives are not boiled. After being used they are carefully washed before being put away. They are sterilized for operation by immersion for twenty minutes in a 1/20 carbolic solution or 3 per cent. formalin. They are removed by a sterile forceps to a tray of 85 per cent. alcohol. This seems to be a safer plan than to depend upon the alcohol tray alone and particularly if the knife has been used previously in a septic operation.

Silk may be prepared by boiling it for ten minutes in a 1/1000 bichloride solution and then for ten minutes in plain water. If the silk is boiled with the instruments to which the soda has been added its tensile strength may be impaired.

The catgut should preferably be obtained from the manufacturer in

sealed glass tubes. These tubes when handled become coated with a thin layer of grease from one's fingers and in this thin film living organisms or spores may lie imbedded and perfectly protected from the action of antiseptic solutions. It is not enough, therefore, to place these tubes in a tray containing carbolic solution or formalin solution. The germs are not destroyed and the nurse, when she takes up the tube in her gloved hands and breaks it protected in gauze, is very likely to contaminate her gloves and as she removes the strand of catgut and draws it out through her fingers she may in turn carry the organisms along the catgut. Infection from the suture may result. This is avoided by boiling the glass tubes of catgut with the instruments. They are then placed in a tray of 1/20 carbolic solution or 3 per cent. formalin.

At the operation the nurse removes a tube of catgut from its tray by using a sterile forceps. She should never put her fingers into the tray. Silkworm gut is boiled with the instruments, also the rubber drainage tubes.

The white enameled basins are sterilized in the utensil sterilizer and a fresh set is used for each operation.

The large glass bowls which are seen in many operating rooms should not be used. There is no reliable way of cleaning them if they become contaminated during a septic operation. The few minutes intervening between one operation and the next do not give the operating-room nurse sufficient opportunity to render them surgically clean.

Mouth guards are worn by the surgeon and his assistants. The speaking voice is capable of projecting minute particles of saliva which carry organisms a distance of three feet, a cough or a sneeze two or three times that far. It seems unlikely that quiet breathing through the nostrils can infect a wound and it hardly is necessary to wear a mask which covers both mouth and nose. As the surgical nurse assists at the operation and sometimes finds it necessary to speak to the surgeon or his assistant, she may speak directly upon the suture which she is holding at the moment only a few inches away from her mouth. It is quite important, therefore, that she also should wear a mouth guard. The same applies to the anæsthetist if the operation is upon the head or neck or shoulders.

And finally it is worth while to mention the very mild infection which may be carried by sweat. It has never seemed to me that this is a serious menace, and yet it is possible that our catgut ligatures or sutures may become infected in this way and prevent the clean healing of the wound.

The sweat may come from the patient's skin as well as from the forearms of the operator or his assistants. No matter how carefully the surface of the patient's skin or the hands, forearms and arms of the operator and his assistants and nurses may be prepared, when the sweat glands begin pouring out their secretion until the sweat collects in droplets there is always a little risk of very mild wound infection.

The climate of Philadelphia is very hot in July and August, and it is not unusual to find ourselves working in an operating room with the tem-

perature near or even above 100° F. The air, furthermore, is saturated with moisture. A leaking skin is inevitable under these conditions. The sleeves of one's gown may become saturated or occasionally a drop of sweat may fall from the gauntlet of one's glove upon the field of operation. Under such uncomfortable conditions, I have found it an advantage to wear a gown with short sleeves and work with gloves and with the arms bare to above the elbows. During the operation I frequently rinse off my gloves and forearms to the elbow in the bichloride basin. The skin of the patient surrounding the immediate field of operation should be frequently mopped with a wet bichloride sponge.

Talcum powder cannot be sterilized in bulk. In the laboratory of the University Hospital we have been able to obtain living spores from the central portion of a box or shaker of talcum powder even though it had been repeatedly "sterilized" in the autoclave. The moist heat cannot penetrate the powder sufficiently to kill the spores which may be lying deeply imbedded in it.

Following is the surgical technic which I outlined for the assistance of our nursing staff in the Orthopædic Department of the University Hospital about two years ago. We have found it satisfactory in every way.

SURGICAL TECHNIC—WARD L.

The Patient.—The day preceding: In the morning give drams two of castor oil, or dram one of aromatic fluidextract of cascara sagrada, and late in the day give a simple enema. In the afternoon prepare the region of operation. This means the whole extremity. In preparing the foot, pay particular attention to the toes, between the toes, the toe-nails, etc. First scrub with tincture of green soap and sterile water—using gauze sponges—for ten minutes. Follow this with sterile water, then scrub and thoroughly douche the part with a warm 1-2000 solution of bichloride of mercury; douche with sterile water and sponge with 85 per cent alcohol. The limb is then covered with dry sterile gauze and bandaged.

The day of operation: A cup of broth or hot milk about 7.30 A. M. Water up to within two hours of operation. On the operating table, the sterile dressings are removed and the limb painted with a 3 per cent. tincture of iodine.

The Operating Room Staff: The surgeon, the assistant surgeon, the resident surgeon. The head nurse, the surgical nurse, the clinic nurse.

The resident surgeon acts as first assistant to the operator.

The assistant surgeon handles the instruments and acts as second assistant.

The head nurse is in the operating room throughout the operation and keeps a general supervision of the nurses and the operating room.

The surgical nurse has charge of the nurses' table and assists at the operation as required. She handles the sterile sheets, sponges, sutures, ligatures, needles, needle-holder, scissors, etc. She never touches anything which is not sterile. She wears a fresh sterile gown for each operation. At the end of an operation she removes her gloves. She disinfects her hands and puts on fresh gloves just before the next operation is to begin. She wears a mouth guard.

The clinic nurse does whatever is required in the operating room where sterile hands are not necessary.

The Operating Room: The washbasin and stands are scoured with "Sapolio" or "Old Dutch Cleanser," or some similar cleansing agent, and the metal parts kept bright with metal polish.

Only white-enamel basins are used in the operating room and these are sterilized in the utensil sterilizer.

On one table there are three basins: No. 1, a warm solution of bichloride of mercury, 1-1000; No. 2, alcohol, 85 per cent., about one-half inch deep and with several gauze pads; No. 3, sterile water.

The bichloride solution should be stained a faint blue, or be marked by a float "Bichloride of Mercury, 1-1000."

On another table is a basin of sterile water which is to be used during the operation. This must always be replaced by a fresh basin for each succeeding operation.

The Nurses' Table: The table is covered with a sterile cloth, and on it are placed:

1. A tray containing packages of rubber gloves of various sizes and the sterile dusting powder.

2. A jar or tray of catgut in glass tubes of various sizes. These may be covered either with a 3 per cent. formalin solution or a 5 per cent. solution of carbolic acid. The tubes when wanted are removed from the jar or tray with sterile forceps.

3. A jar containing silk of different sizes and kept the same as above.

4. A jar of rubber drainage tubes, and kept the same as above.

For each operation the surgical nurse spreads a fresh sterile sheet or cloth across the front half of this table, and upon this she places the fresh sterile tray which contains the scissors, needle-holder, needles; and a glass tray which contains the scalpels and tenotomes covered with alcohol. The nurse touches nothing on this table except the two trays and their contents and the sterile sheet upon which they rest. She must be careful not to contaminate the contents of any of the glass jars or trays which contain the catgut tubes, etc., but must always remove what she requires with sterile forceps. These forceps are, of course, re-sterilized with the other instruments before the next operation.

At the close of the operation, then the two trays and the sterile sheet are removed, to be replaced by fresh ones for the next operation.

Nothing which may have come in contact with one operation—either directly or indirectly—is to be permitted to come in contact with the following operation, either directly or indirectly.

The Instrument Table: The instrument table should be covered with a sterile rubber cover and over this a sterile sheet, and upon this are arranged only the instruments which are required for the operation. At the end of each operation *everything* is removed from this table.

Sterilization.—The gauze dressings, gauze sponges, towels, sheets, gowns, etc., are sterilized in the autoclave by live steam, followed by dry heat. Each operation is to have its own separate drum. One drum may be used solely for gowns of nurses and doctors, and this may remain in its position on its stand throughout the series of operations.

The instruments (which include everything except the knives) are sterilized by boiling for ten minutes.

The knives and tenotomes are covered with a 3 per cent. solution of formalin for 20 minutes. This is poured back into the stock bottle and the knives are covered with alcohol until used. The tray which is used is sterilized in the utensil sterilizer.

The Rubber Gloves.—These are washed with soap and water to remove all stains, turned inside out and washed again. They are then filled with water and put into a vessel of boiling water with a wire frame on top of them, so that they cannot float up on top of the water. They are boiled 5 minutes by the watch. When the water has cooled, the nurse, wearing sterile rubber gloves, removes the gloves from the water and dries them with a sterile towel, of course turning them inside out in doing this. The glove is dusted inside and out with sterile powder and folded in a piece of sterile muslin and made into a packet. These are then placed in a large glass jar having a glass cover, and put away until they are needed for operation. They are then put

into the autoclave and sterilized with the surgical dressings during their final sterilization. The gloves need not be left in the autoclave longer than 20 minutes.

The glass tubes of catgut may be sterilized with the instruments and then placed in carbolic or formalin solution.

Drainage tubes should be boiled 10 minutes and then placed in the formalin or carbolic solution.

Silk, if it is to be left buried in the tissues, should be boiled for 10 minutes in a 1-1000 bichloride solution, and then for 10 minutes in water.

INSTRUMENTS REQUIRED

For every operation have ready the following:

2 pairs of retractors
2 scalpels
2 tenotomes—1 sharp-pointed and 1 blunt
12 hæmostats
4 curved hæmostats
4 tenaculum forceps
4 Allis forceps
2 dissecting forceps
2 rat-tooth
2 pairs scissors, blunt ends—1 pair curved and 1 pair straight
1 Allis dry dissector
1 grooved director
1 small probe
1 periosteal elevator
1 needle holder
needles
1 ligature carrier

Additional instruments are required for certain operations as follows:

Tendon transplantation:
- 1 long, very slender pair of forceps
- 1 long probe with eye
- 1 drill handle and 3 drills
- 1 very narrow osteotome

Arthrodesis and transverse horizontal section:
- All the preceding instruments and also
- 1 medium gouge
- 1 small gouge
- 1 medium chisel
- 1 small chisel

Bone grafting:
- 1 Hey's saw
- 1 osteotome—medium
- 1 osteotome—small
- 1 chisel
- 1 mallet or hammer
- 1 bone-cutting forceps

Osteotomy:
- 1 osteotome—large
- 1 osteotome—small
- 1 osteotome—medium
- 1 hammer or mallet
- 1 König block
- 1 sand pillow

Arthroplasty:
- 2 saws
- 2 chisels
- 2 gouges
- 1 curette
- 1 bone-cutting forceps
- 1 hammer or mallet
- 1 drill
- 8 extra hæmostats

Erasion of a joint:
- Same as Arthroplasty and
- 1 lion-jaw forceps
- 1 sequestrum forceps

Talcum powder cannot be sterilized if it is in bulk; that is, in the dusting can. The talcum powder should be spread on a gauze sponge in a very thin layer and then placed on top of the rubber gloves just before they are enclosed in the muslin covering.

EDITORIAL COMMENT

WAR SURGERY OF THE ABDOMEN

CUTHBERT WALLACE, Consulting Surgeon of the British Armies in France, has published in an octavo volume of 152 pages (Philadelphia: P. Blakiston's Son & Co.), a study of the experiences during a period of thirty months extending from July 1, 1915, to December 31, 1917, in abdominal surgery of the sector of the battle line under his surgical control.

The number of cases upon which the study is founded is 1098 abdominal wounds. The object of the study is to determine what benefit may be expected in dealing with these cases by operative means. All cases occurring within the particular sector involved are included, no matter at what hospital they are treated. It goes without saying that the results are greatly influenced in different hospitals by their respective distances from the firing line. They differ greatly, also, according to the nature of the fighting. They are nearly always worse at the end of a fight than at the beginning, for the cases at the end are difficult to collect, and are, therefore, late in arriving. Again, if times are quiet, there is plenty of time to treat all cases adequately and deliberately, the conditions being quite different from those in which active operations are in progress, when it may be only possible to pay attention to the most favorable cases. It is necessary, also, not only to reckon the mortality following operations, but also the mortality in those cases in which no operation was performed. The statistics obtained are the result of a system whereby every hospital which was called upon to treat abdominal cases was provided with a book in which certain headings were written down, which were filled in at the time of operation by the medical officers.

In the earlier months of the recent war an expectant method of treatment was generally adopted. Operations were secondary or late. A wounded man was first received by his regimental medical officer to whom he was brought in the regimental aid post, situated somewhere in the trench system; thence he would be transferred by stretcher to an advanced dressing station, usually situated just behind the trenches; thence by car to the field ambulance, where he was kept until the arrival of a motor convoy (often only once in twenty-four hours), by which he was taken to the casualty clearing station, which was the first place adequately equipped for operative treatment. Here there was reasonable possibility for the performance of any operation thought advisable. In some cases, with the idea of avoiding a disturbance by movement, a man wounded in the abdomen was kept at the regimental aid post; often he was kept at the field ambulance, but usually he was transferred to the casualty clearing station as soon as possible. Here the usual procedure was to put the man in the Fowler position; to improve the general condition by rest and warmth; to withhold food and water for three days, and to administer morphia. Thirst was combated by rectal salines and mouth-washes. The result was that many patients at first gravely ill went through a period of improvement which often was very striking, so that it was possible to evacuate them to the base hospital. Here they arrived sometimes in good condition and sometimes gravely ill; as a rule, they became worse and worse, and in the end died.

In 1915 a series of operations and post-mortem examinations showed that the intra-abdominal injuries were of such a nature that recovery was not to be expected without surgical aid except in a few instances.

Hemorrhage was a frequent cause of early death. The injuries produced by bullets often were very extensive, negativing the prevalent opinion as to the comparative smallness of the lesions produced by a modern small-bore bullet. Appreciation of the large part which hemorrhage played in causing the early death after such

injuries emphasized the necessity of the earliest possible operative interference for its control. Directions to use all possible celerity in forwarding all abdominal wounds to the nearest casualty clearing station were followed by rapid improvement in the results obtained.

With regard to the relative frequency of abdominal wounds, experience in the present war has been that the number of such wounds that reach an operating hospital is not likely to exceed 2 per cent. of the total wounded received, provided no segregation of such cases is practiced.

It has always been a much-debated point as to whether a projectile could traverse the peritoneal cavity without wounding the hollow viscera. The result of the large number of cases in which, after perforating wounds, the abdomen was opened and explored, is that in a number of them no hollow viscus was found injured, although the abdominal cavity was usually full of blood. In some of them the injury was limited to bruises and tears of the peritoneal and muscular walls of the stomach or intestine. It is not denied that in rare instances spontaneous recovery after wounds of the hollow viscera has occurred. Wallace gives the details of a number of such instances, but the number is too small to have any practical bearing upon the indications for treatment in any given case.

The relative frequency in which the different organs of the abdomen are wounded, from a total of 965 cases, is as follows: Small intestine, 363; colon, 252; liver, 163; stomach, 82; kidney, 74; spleen, 54; bladder, 45; rectum, 21; pancreas, 5; ureter, 3.

Most cases succeeded in reaching the casualty clearing station some time between six and ten hours after receipt of the injury. The statement is made that up to six hours the chances are in favor of the patients; after this period they are always against them. So far, the limit in successful cases has been forty-eight hours for resection of the small intestine and thirty-six hours for a suture of the colon and for a suture of the stomach.

Notwithstanding the realization of the fact that the sooner a man is operated upon the better, there is another factor, namely, the aftercare of the wounded man. He must be kept quiet and well nursed. While it might be sometimes physically possible to operate upon him at the regimental aid post or advanced dressing station, it would be under the most disadvantageous circumstances. After operation, nursing as usually understood would be impossible. In addition, the man would be under constant shellfire or close to our own guns. To avoid delay in such and other cases of severe wounds, the advancement of the casualty clearing station reasonably far forward is important. In some instances it has been possible to establish special hospitals in advanced positions.

With reference to abdominal wounds, the author states that the French and the Belgians have gone through the same experience as the British. Since the fighting line became fixed, operation has been the accepted practice. Advanced abdominal hospitals have been established at some centres.

As regards the diagnosis of intra-abdominal damage, experience has shown that one has to be very careful in making a negative diagnosis, and it has also shown the wisdom of operation in doubtful cases. Though a man hit in the abdomen usually looks ill, he sometimes appears to have no serious lesion. Sometimes he is apathetic and quiet, sometimes restless. He may be blanched, or of a fair, clear color, even normal in appearance. He may present a good facial expression, even though his extremities are cold and clammy, and the pulse imperceptible, and death imminent.

When the patient has once arrived at an operating hospital, it is better to put the man to bed and watch his condition for a time rather than to immediately subject him to operation. The first and foremost indication is to secure the warming of the patient. This is by far the best treatment of shock, and greatly excels in value the administration of stimulants of whatever nature. If heat, rest and morphia do not improve the man's condition, it is doubtful if transfusion or infusion will produce

any effect, except in cases of severe hemorrhage, when transfusion of blood is by far the best restorative.

The actual moment for performing the operation must be left to the individual judgment of the operator. If the man does not respond at all, the question must be decided whether it is best to operate at once, or whether it is worth operating at all. Frequently some hours will produce such improvement that an operation is possible, where at first the case seemed hopeless. Experience has shown the wisdom of operating as a routine measure. The main question is, What case is it best not to operate?

It is true, also, that there will be some patients operated upon who are not found to have any visceral injury, and some in whom the wounded parenchyma of solid organs has ceased to bleed.

There are, of course, many cases in which no surgeon would feel himself justified in operating, but there are many borderline cases which some surgeons would leave and others would feel constrained to submit to operation. The bolder surgeon may get the worse operative mortality, and yet save more lives.

Wallace accepts twenty-four hours as the usual limit within which a primary operation is likely to be successful. By this time hemorrhage will have ceased, and operation is only to spread infection, if the bowel has been perforated, and to hasten the end.

With reference to wounds of particular organs, out of 965 cases operated upon, the stomach was perforated 82 times, in 55 of which it was the only hollow viscus damaged. In cases uncomplicated with wounds of other hollow viscera, the mortality was 52.7 per cent. The small intestine in this same series of operative cases was found wounded 363 times, in 255 of which it was the only hollow viscus wounded.

Single lesions are the exception. Out of 124 cases, the average number of lesions was 4.8. The greatest number of lesions was 20. The lesions are usually collected close together, but sometimes they are disposed in two or three groups, which necessitate multiple resections.

As a rule, the whole length of the small intestine must be explored. Suture should be practiced when possible. Small discrete injuries, however numerous, should be sutured. It is also better to narrow the bowel than to resect, even if it involves a lateral anastomosis. Resection should be reserved for cases where the bowel is practically destroyed, where there are several severe injuries or complete divisions close together, and where injuries extending into the mesentery or infarction of vessels give no choice. A single row of stitches is practically all-sufficient in the suture.

In the 965 operative cases under consideration, the colon was wounded 252 times. In 155 cases it was the only part of the alimentary tract hit. The injuries of the large intestine are similar to those of the small gut. The differences that are to be noted are due to its larger size and the absence of coils, to the absence of mesentery in portions of its course, and its proximity in some portions of its extent to bone.

With regard to the treatment of colon cases, the author is of the opinion that, although some unoperated colon cases did recover, nevertheless these cases cannot now be advanced as a ground for abstention from operation except in cases which arrive late.

The mortality of colon cases uncomplicated with other lesions of the alimentary tube is 58.7 per cent. Where the simple suture has been possible, the mortality was only just above 50 per cent. When a colon anus was required, the mortality was 73.5 per cent. This increase in mortality in cases of colostomy is due not to the character of the operation itself, but to the fact that progressive sepsis was not prevented from infection already started when the colostomy was performed.

The bladder was perforated 45 times in the 965 operated cases, and in 25 cases was the only organ injured. Of these 25 cases, 14 died.

Shock and hemorrhage form the causes of death in the majority of cases.

In addition to the abdominal injuries, every kind of bodily injury may also be present in individual cases. Compound depressed fractures of the skull, fractures of the long bones, multiple wounds, fractured spine, avulsed limbs, are among such complicated injuries, but for such extraneous injuries the mortality in the cases studied would be considerably lower.

Hemorrhage stands out as the great source of danger. In wounds of the stomach, damage to the vessels running to the stomach wall before they penetrate it and to the big vessels lying on the two curvatures may give rise to very great hemorrhage. The omental vessels may be a source of hemorrhage. The small intestine wall is the source of considerable hemorrhage. The mesenteric vessels give rise to very considerable hemorrhage. A mesenteric wound often includes also a wound of the big vessels, such as the colics, and it is the most common source of blood in the abdomen when cases come to operation.

Hemorrhage in the retroperitoneal tissue is a source of great trouble. The formation of extensive hæmatoma is common. The deep epigastric artery of the abdominal wall may be the source of hemorrhage. The great intra-abdominal vessels may be injured. The parenchyma of all solid organs bleeds freely at first; but if no large vessel is injured, the hemorrhage may be expected to cease spontaneously in a few hours.

Peritonitis is the usual cause of death after the period of shock and hemorrhage is over. The amount of peritonitis present when an operation is done at an early stage varies much. It is not so dependent upon the time elapsed since the wound as might be expected. Infection may be due to contamination from the bowel, to dirt carried in by the projectile, and, finally, from the operation wound itself from fault in technic.

There are many other points of detail which are discussed in this little book. Every page is full of interest, being a study on a large scale of a vast experience—experience which was possible to be gained only under the conditions of a great war such as that which has been waged during the past four years and more. The abrupt closure of the war has made its lessons less pressingly important upon the clinical side. As one reads the details of the conditions in which much of the work here recorded has been done, one is filled with admiration of the high spirit of devotion with which the surgeons of the Allied forces have applied themselves to their work.

Every one will gladly adopt the language of the author when he pays compliment also to those who collected and bore the wounded from the field to points of relief. What success the surgeons have obtained, he says, is due mainly to the magnificent way in which the wounded have been collected by the bearers. One cannot sufficiently express one's admiration for their courage, steadfastness and endurance. The motor ambulances of the field ambulances and of the motor convoys deserve great credit. What praise is not due to the drivers, who, in darkness and shellfire, tenderly picked their way among the holes in the road so as to avoid a needless jolt to their wounded charges? The book as a whole is fittingly dedicated to *"The Stretcher-Bearer."*

THE LATE RESULTS OF GUNSHOT WOUNDS OF THE CHEST

As an addition to the contributions to the immediate surgery of wounds of the chest which were the subject of editorial comment in the November issue of the ANNALS OF SURGERY (pp. 554–562), interest will attach to contributions upon the late sequelæ to such wounds, which form a series of papers published in the *Lyon Chirurgical* for May–June, 1918.

In the first paper (*Récherches cliniques et radioscopiques sur certaines sequelles lointaines des plaies pleuro-pulmonaires de guerre)*, the authors, MM. Pehu and Daguet,

deal with certain consequences found present several months after the traumatism had been sustained and when every evidence of bony, bronchial, pulmonary, or pleural disturbance of a suppurative or inflammatory nature had ended; when cicatrization had been accomplished, but when in most of the cases there could still be observed a tendency to slow spontaneous betterment, the cicatricial condition not yet having become definitely fixed. Various recent papers have treated upon the general subject of the later consequences of thoracic wounds. They show that, to the acute immediate phase succeeds a period during which, at least in a certain number of them, it is quite frequently the fact that subacute manifestations may be found showing that the process is not fully and completely terminated. The authors of the present paper have studied such sequelæ of thoracic wounds with the special idea of contrasting results in those in which there were present sero-fibrinous effusions of infectious origin, with those which were bloody and purulent in their nature from whatever origin they may have sprung. In all the cases studied by them, several months (four at least) had intervened between the reception of the wounds and the time they came under the authors' observation. The acute phenomena had ended, pleural suppuration had ceased, and the bronchial manifestations had been suppressed.

The problem was then to estimate the physical value of the individual as to final restoration to health, or as to the necessity for excluding him from the list of possible future combatants. The material was composed of soldiers of from 20 to 40 years of age belonging to all classes of fighters. Of these they have examined in all 146 in the space of twenty months.

As to the nature of the missiles which had inflicted the wounds, shell fragments were much the most frequent, being from 85 to 90 per cent. The rest were rifle balls, or fragments of shrapnel or hand grenades, or similar agents.

Out of the entire number there was but one wound from a revolver ball, and one dagger wound.

As to the clinical symptomatology presented in these cases, the physical signs are identical with those in chronic pleurisy from whatever cause. The symptoms are in general not well marked; a dulness scarcely appreciable, or an ill-defined diminution of the respiratory murmur. One does not note the frank dulness which accompanies certain forms of parietal adhesions, especially at the base. Auscultation reveals a varying degree of respiratory suppression. As to fremitus, it is always appreciable to a degree more or less pronounced. A phenomenon quite constant, noted even in the least severe cases, consists in the diminution of expansion of the half of the thorax corresponding to the wound. This sign is not sought for as often as it ought to be. It possesses a very great value in directing research. It is noteworthy that there does not exist a close parallelism between the data furnished by the X-ray and the mobility of the corresponding half of the diaphragm. A thorax may present the least degree of mobility while the muscular curtain is but little paralyzed. Inversely, certain thoracic frames may dilate to a sufficient degree while the diaphragm is almost inert. This is due to the supplementary activity, more or less efficient, of the accessory respiratory muscles.

Besides these particulars revealed by physical examination of the thorax, signs of persistent bronchitis or chronic pleurisy have been rarely noted. Generally, it may be said that the objective physical signs referable to the framework of the thorax are always less characteristic, more imperfect, than the signs revealed by the X-ray. The signs obtained by radiographic examinations merit careful study. They show exactly the functional value of the pleuro-pulmonary apparatus and serve as valuable guides in an estimation of the later course of the case.

In a certain number of cases the presence of metallic foreign bodies was shown, situated in various portions of the thoracic cavity, of which, in the opinion of surgeons, extraction was judged useless or dangerous. Of the 146 cases under examination, there were 27 bearers of intrathoracic projectiles. These foreign bodies were apparently well tolerated and around them there could be seen no modification of the radioscopic

image of the intrathoracic viscera. In one case only was noted the existence of a grayish halo around a foreign body.

The clinical signs, therefore, as far as the sequelæ of wounds of the chest are concerned, are of much less importance than the signs revealed by radioscopic examination. The fluoroscope screen shows often conditions unsuspected by external investigation alone, and to arrive at an exact conclusion as to the anatomical and physiological characteristics of the ultimate results radioscopy is essential. The screen examination makes possible the determination of the degree of alteration of the diaphragm, of the pleural culs-de-sac, of the pleura itself, etc.

The authors have divided the thoracic wounds studied into two classes: (*a*) Those which have been accompanied by a purulent pleurisy subjected to incision, and (*b*) those which have not undergone any surgical intervention. The sequelæ consecutive to wounds complicated with empyema do not entail changes much pronounced and very extensive. Most frequently may be observed shadows localized along the sinus; a slight thickening is at the base of the lung, a little diaphragmatic inertia betraying itself to external examination by the least signs of chronic pleurisy. One was often surprised to find that empyemata which had suppurated for many months, left behind them slight signs; that the play of the diaphragmatic dome is very little altered; that the stroma of the lung is scarcely rendered grayish. Even if the incision of the purulent collection has been more or less delayed; if, according to all probability, it has been preceded by a period of simple hæmothorax, nevertheless, the remnants left are minimal.

The most favorable conditions, the minimum of sequelæ, have resulted from interventions in which pleurotomy had been accompanied by costal resection. Rib resection is indispensable to permit sufficient drainage through the rubber tubing introduced more or less deeply into the chest. Division of the skin, muscular planes, and incision of the pleura without touching the ribs is not sufficient.

As to the wounded who present no operative cicatrix, a considerable number of the penetrating wounds, whether accompanied or not with bloody effusions, left no important sequelæ.

A special study has been given to the frequency of the development of pleuropulmonary tuberculosis as a sequelæ to the injuries of the chest. Of the 146 cases under examination there were observed three cases of pulmonary tuberculosis and two cases of sero-fibrinous pleuritis of an undoubtedly tuberculous nature. It was impossible to say positively, however, whether these cases were dependent upon the injury received or not.

THE EARLY TREATMENT OF SEPTIC PLEURAL EFFUSIONS COMPLICATING PENETRATING WOUNDS OF THE CHEST

(Note sur le traitement précoce des épanchements septiques de la plevra, complications des plaies pénétrantes de poitrine.)

Messrs. V. Combier and J. Hertz (Aide majors à l'Ambulance automobile 13) report their results in the early closing of the thoracotomy wound in cases of septic pleural effusions as soon as the pleural cavity had been rendered sterile by drainage accompanied by irrigations after the method of Dakin. According to their experiences, sterilization of the pleural cavity can be accomplished very rapidly if it is begun early. The objectives to be attained were to empty and disinfect the pleura, to close it, and to mobilize the lung.

Only in cases in which a wounded man retained within the thorax a large missile likely to be poorly tolerated, and especially likely to reinfect the wound, did they practice the extraction of the missile, if it did not necessitate deep penetration into the lung. In this course they were influenced by the theory that the extraction of an intrapulmonary projectile was less grave in its consequences when the inflammatory process has disappeared. Again, whenever the cause of pleural infection could be

traced to an infected focus attending fracture of the ribs, they excised this complicating focus. If the focus is a fracture of the scapula, its clearing out is still more necessary on account of the muscular masses which surround it. The presence of a fracture of the rib in a chest wound which it continually aggravates is of itself alone an operative indication after the arrival of the patient at the hospital.

The time for surgical intervention has in all cases been controlled by bacteriological and cytological examinations made by the chief of the laboratory service. The results of these examinations have served as controls when compared with the later examinations practiced upon the pleural fluids after the Dakin irrigations. These examinations have furnished information as to the transformation of the pus and its flora, the progressive diminution of the number of germs.

In all cases the closure of the wound was done only when the number of microbic elements had become very minute, and when the percentage of white corpuscles showed that the proportion of the poly- and mononuclears had become normal.

Operative Technic.—It is supposed that, before any intervention, X-ray examination has been done to secure information as to the quantity of intraperitoneal liquid, as to the presence of the projectile, or of fragments of the ribs. The authors have always employed by preference local anæsthesia, with 1 per cent. solution of adrenalized novocain, accompanied by one or two hypodermic injections of morphin, one at the beginning of the local anæsthesia and a second in the course of the intervention, if it seemed necessary. An important effect of such hypodermics of morphin the authors believe to be the prevention of reflex nervous accidents due to spasm of the cerebral arteries with transitory ischemia, which the production of the pneumothorax may entail.

The first step is the resection of some cubic centimetres of the ninth rib in the posterior axillary line, preceded by an exploratory puncture at this level to make sure that the drainage would be effectual. Once the rib has been resected, they make a pleural incision, which is begun with the point of the bistoury and prolonged progressively. In the case of thoracotomy for purulent pleurisy, the ingress of the air into the pleural cavity is little to be feared. The wounded lung is already prepared by the previous progressive compression due to the effusion. The thoracic equilibrium has already been broken and it would not be subjected to the three great causes which explain the accidents due to the opening of the pleura, namely, deviation of the mediastinal diaphragm, air embolism in the circulation, the production of pneumothorax. After the liquid has been evacuated and direct finger examination has been made, the careful removal of false membrane is instituted. Inspection of the cavity to make certain that it is free from retained masses may be made by suitable light. Then they put in place the irrigating tube and the drainage tube. The latter descends into a reservoir containing at its bottom liquid which may produce suction to keep the pleura dry and to draw the lung to the wall of the chest. This pleural drainage tube is of the calibre of the index finger. It should penetrate into the pleura five good centimetres; it should have three large, long fenestra; it should be extremely soft, so that it may cause no irritation. Into its large lumen may engage clots and false membranes. It is fixed to the skin by a point of silkworm gut.

The irrigating tube is a long tube which, in the majority of cases, passes up into the pleural cavity through the operative wound 20 centimetres, toward the summit, where it is fixed by a point of silkworm-gut suture.

In cases of recent pleurisy, or those in which diverticula are absent, one irrigating tube may be used instead of the multiple tubes which have been recommended. By several points of suture involving the pleural muscles and skin the wound is hermetically closed about the tubes. Upon the closed operative wound through which emerge the two tubes two long compresses are placed and the whole is covered over by a plate of pure rubber, perforated by two openings, through which come out the two tubes, the drainage tube going to its bottle, the irrigating tube fixed to the shoulders and kept closed between the times of injection. The circumference of the rubber

plaque is fixed to the skin by cement. By means of this arrangement all the pleural liquid, pus and Dakin fluid passes into the drainage bottle, the bed is not soiled and reinfection from the wound is rendered impossble. It is sufficient after three days to change the two compresses which rest upon the skin. The patient is put in bed in the sitting position. The end of the drainage tube is placed in a bottle filled one-third full of antiseptic liquid. During the first day there are made five instillations of 20 cc. of Dakin fluid; two more during the night; the same again the second day, with the addition of one transpleural irrigation of from 250 to 500 cc. of the Dakin fluid at one time. This is continued to the fourth or fifth day, when the number of instillations is reduced to four, of from 20 to 50 cc. each day. Never has it been found that these irrigations caused functional troubles. The patient bears them readily and often expresses a feeling of comfort from their use. The drainage tube rarely comes out. If by chance the flow is interrupted, it soon is restored, the Dakin solution having broken up the fibrinous masses which obstructed it.

When the temperature has completely fallen to normal and no longer rises again, a condition which, when the treatment has been early instituted, may in general be secured by the fourth day, when the general state is clearly improved and one can see a radical change for the better from day to day; finally, when the laboratory examinations permit it, the two tubes are taken away and the total closure of the wound is made by sutures, which involve the entire thickness of the wall and pass as near as possible to the pleural edges. Precaution should be taken to withdraw the two tubes and to put the sutures in place while the patient is in a condition of forced inspiration, nose and mouth closed, so that the lung being brought up to the thorax wall may limit as much as possible the pneumo-thorax of the disinfected cavity. The day after the closure a spirometer is given to the patient, who begins to make respiratory gymnastics, with the object on one side to dilate the lung and on the other to prevent adhesions to the wall of the thorax.

Although the authors never sought the primary extraction of the projectile in these infected pleuræ, nevertheless in five cases they were forced to undertake it. The projectile had been previously seen and localized by the X-ray, and they were led to it through a focus of rib fracture, or through a retroparietal abscess opening at its depth in the pulmonary cavity where the missile was found, either as it had been quite easily felt or in the midst of adhesions or false membranes. In two cases the projectile was found in contact with the wounded pleura.

In searching for these foreign bodies they were greatly helped by the spreader of Tuffier. In five cases the projectile was removed in the course of the thoracotomy for the pleural suppuration. In four cases they made removals of splinters or resection of the ribs. In thirteen cases they had to do with wounds produced by shell fragments. In all, fifteen cases are detailed, with one death, the patient dying from double pneumonia on the second day after infection.

The analysis of these cases demonstrated that the method of early closing of thoracotomy wounds in cases of purulent pleurisy does not involve danger for the patient. Should the temperature again rise from retained infected material after the closure, it would suffice to make exploratory punctures, and, if they were positive in the result, to open the operative wound, to drain anew and to replace the disinfecting tubes.

The results obtained by this method were excellent. Upon an average, at the end of three weeks examination showed the thoracic wall not sunken in, lung mobile and painless, without signs of pleurisy and with normal breathing over the whole lung. The wound of the patients thus treated was closed at the end of fourteen days and healed permanently. Others were subjected to disinfection during periods varying from fifteen to sixteen days. Many of their colleagues had applied the same technic, with the same results.

TOTAL PLEURECTOMY FOR PACHYPLEURITIS

(Quatre nouveaux cas de pleurectomie totale pour infection pleurale avec pachypleurite.) Dr. J. L. Roux-Berger reports four cases of pleurectomy with decortication of the lung done for the pachy-pleuritis due to pleural infections consecutive to wounds of the lung from shell fragments. In these four cases the infection evidently had as its cause incompleteness of the original operation. The orifice of entrance of the fractured rib had been correctly treated in the primary operation, but the shell fragment within or near the pleura had been left behind.

As to the technic of the pleurectomy, the difficulty consists, once the lung has been released, to assure a sufficient drainage of the pleura without such drainage being too prolonged so as to prevent a new retraction of the lung and the formation of a new sac immobilizing it.

The operative consequences were of the most simple character. The most rapid cure was obtained in a patient in which a very extensive pneumopexy was done, fixing to the parietes almost the entire lower border of the lung, and thus preventing secondary retraction. The author purposes, if he should have to treat another similar case, to attempt by extensive pneumopexy to fasten to the parietes a very large surface of the lung tissue. As far as the distensibility of the lung will permit it, such a manner of treatment will insure all the advantages of drainage without its inconvenience.

Of the four cases subjected to operation, three were discharged cured; that is to say, they were completely healed, but it is not claimed that this anatomical cure corresponds to a physiological cure. In the fourth case a small pleural fistula remained.

Lungs immobilized for so long a time within a rigid case in contact with a suppurating cavity fixed to the thoracic wall by adhesions, can only regain a very mediocre functional ability.

All the patients came under treatment in very bad condition with suppurating fistulæ; in two of them bronchial fistulæ were present. In two of the patients the operation was divided into two steps, with intervening periods of from three to six weeks. In the first intervention a large costal resection was done, with excision of the fistula, removal of the projectile, the cutting away of the thickened part of the parietal pleura, the careful closing of the whole pleural cavity, with regular drainage and irrigations after the method of Dakin until the cavity had become sterilized. At the second intervention, after the removal of the newly formed osteo-fibrinous tissue, the lung was decorticated by excision as completely as possible of the thick fibrinous cover which bound it down. The inflated lung was then fixed to the chest wall by various points of suture. The wound in the chest wall was then sutured, with a drain in place.

The author advocates in similar cases as extensive a pneumopexy as possible. The broader the fixation the better the result.

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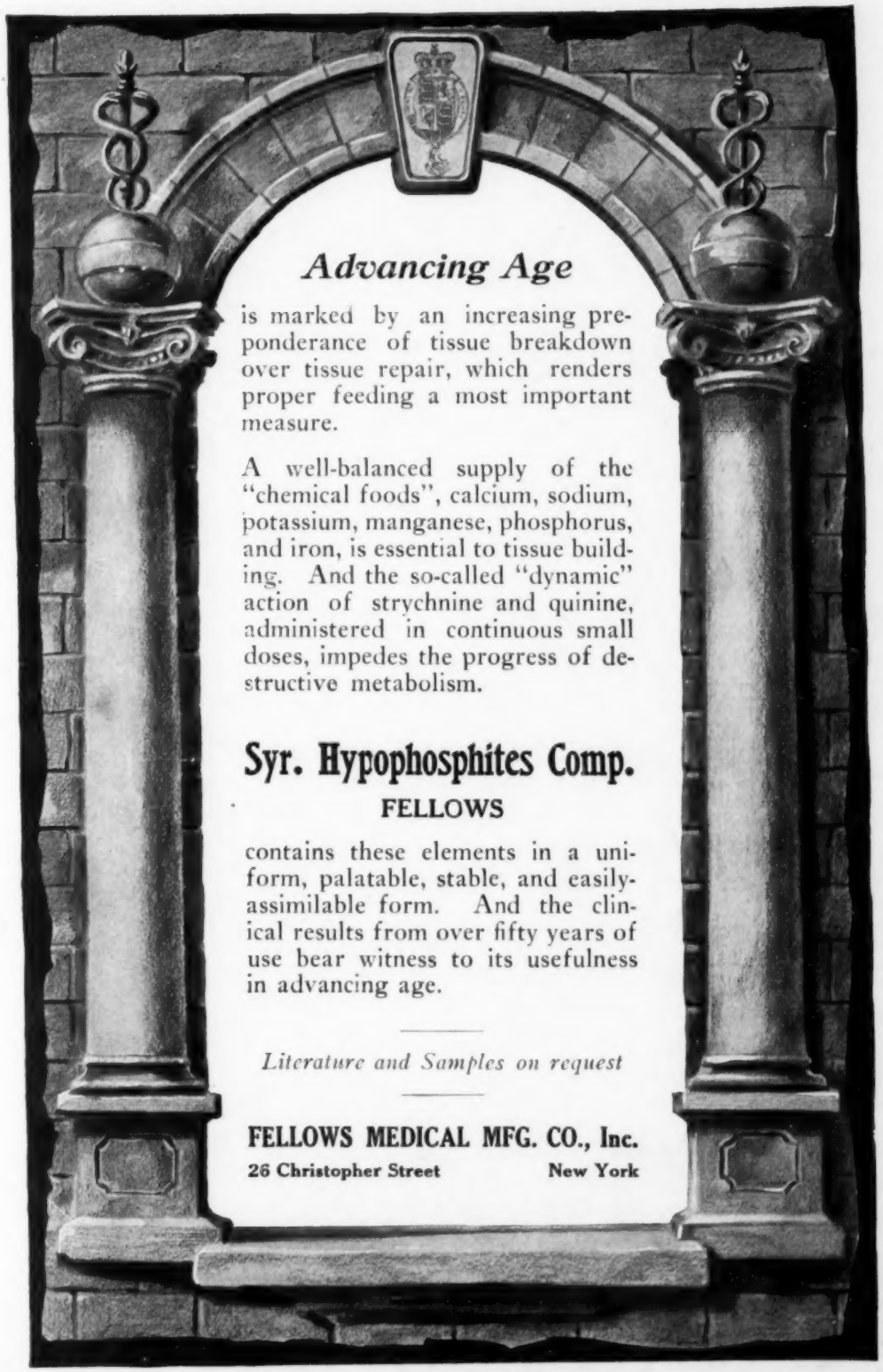

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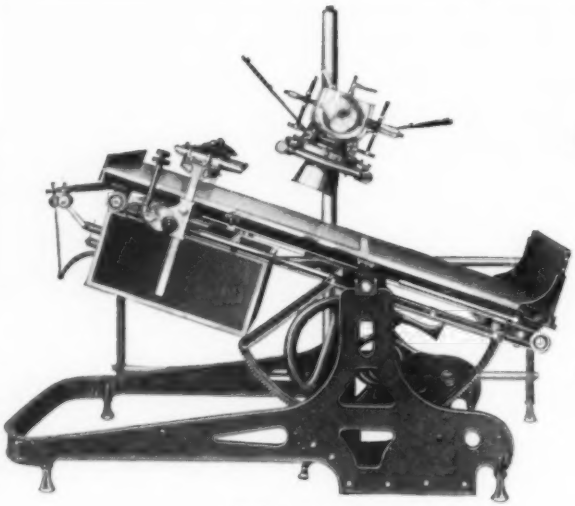

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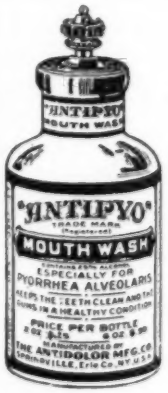

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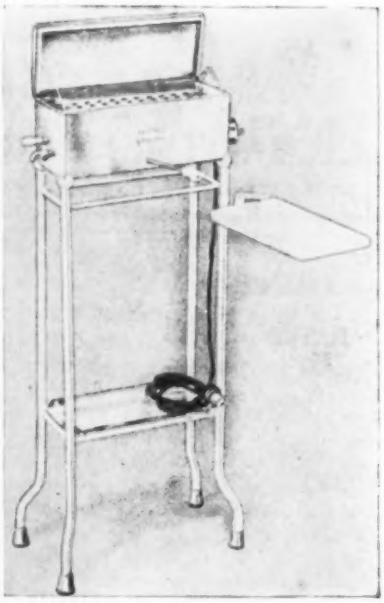

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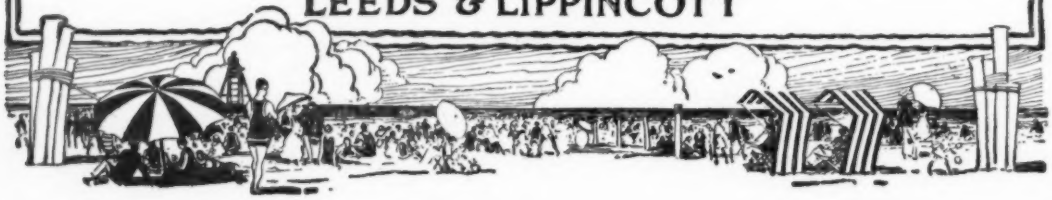

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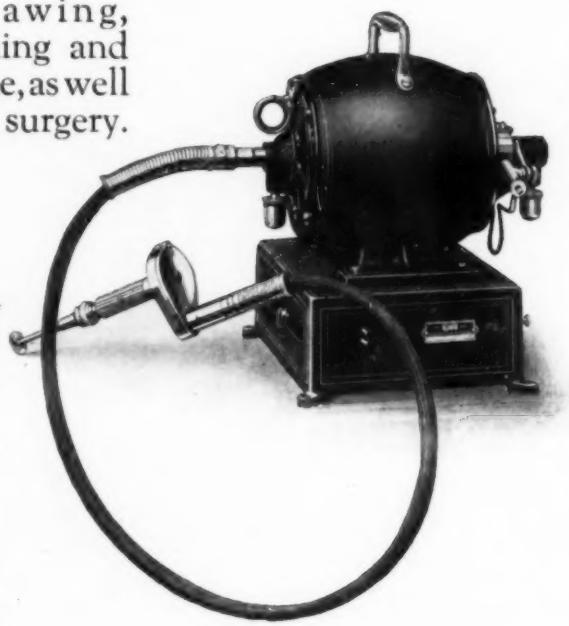

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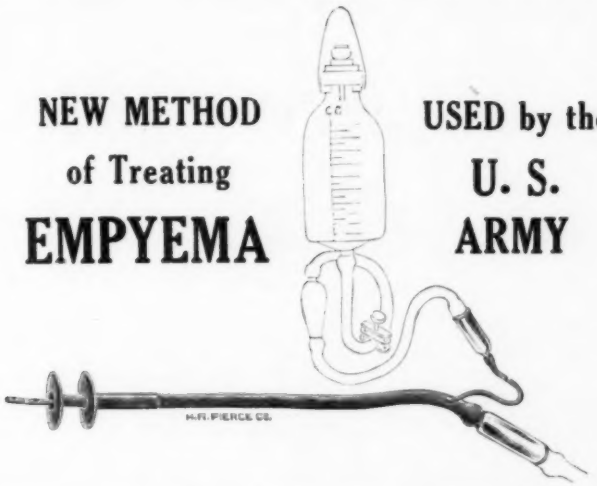

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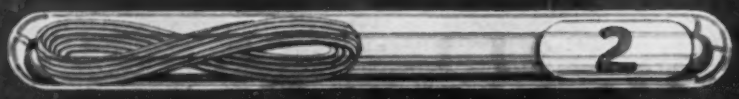

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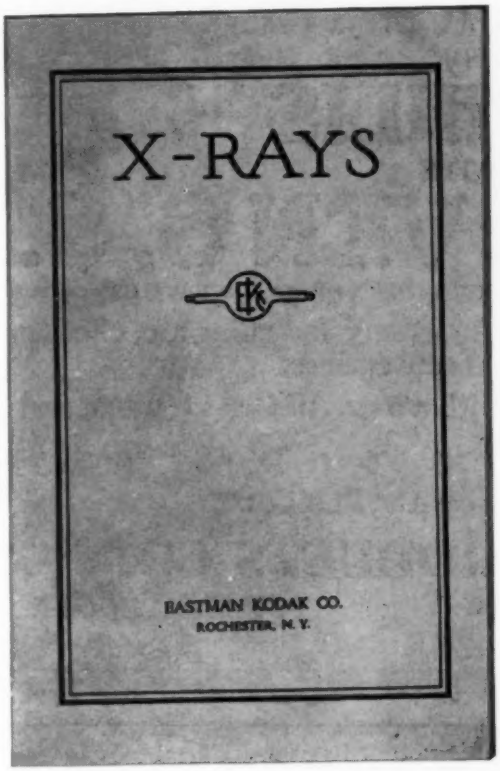

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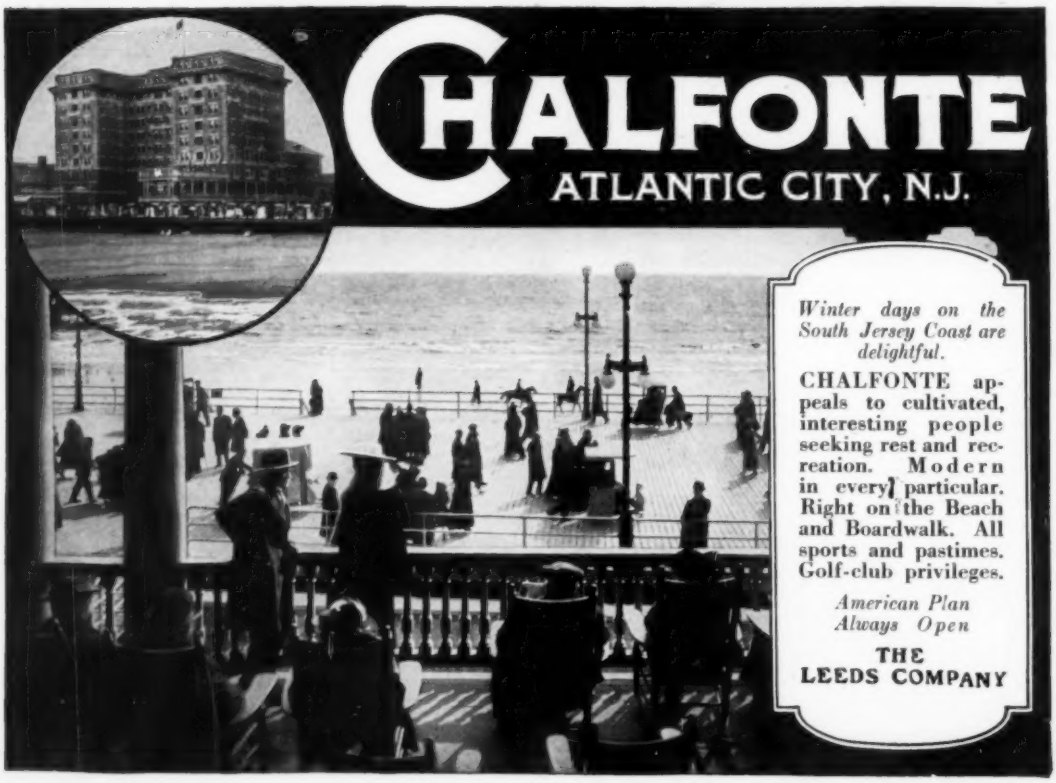
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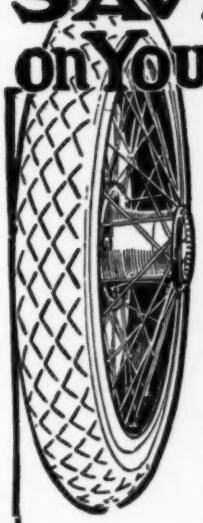

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